A DISSERTATION ON

"A COMPARATIVE STUDY ABOUT THE EFFECT OF DEXMEDETOMIDINE VERSUS LIGNOCAINE ON HEMODYNAMIC AND RECOVERY RESPONSES DURING TRACHEAL EXTUBATION"

IN COIMBATORE MEDICAL COLLEGE HOSPITAL

Dissertation submitted to

THE TAMIL NADU DR. MGR. MEDICAL UNIVERSITY,

CHENNAI – 600032. TAMIL NADU.

In partial fulfillment of the regulations required

For the award of the degree of

M.D. DEGREE BRANCH-X

ANAESTHESIOLOGY

GOVERNMENT MEDICAL COLLEGE AND HOSPITAL,

COIMBATORE, TAMILNADU.

APRIL 2016

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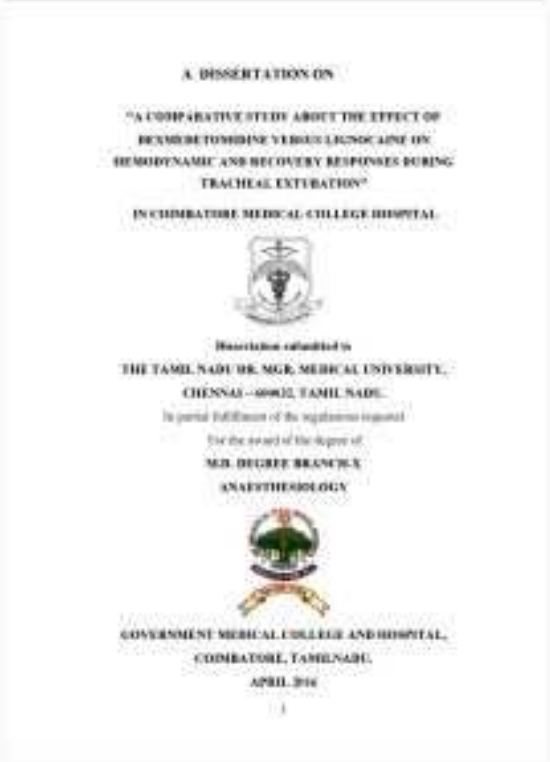

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CERTIFICATE

This is to certify that this dissertation entitled **"A COMPARATIVE STUDY ABOUT THE EFFECT OF DEXMEDETOMIDINE VERSUS LIGNOCAINE ON HEMODYNAMIC AND RECOVERY RESPONSES DURING TRACHEAL EXTUBATION"** submitted by **Dr. J. LAKSHMI** in partial fulfillment of the requirement for the degree of Doctor of Medicine in Anaesthesiology by the Tamilnadu Dr .M.G.R. Medical University, Chennai is a bonafide record of the work done by her in the Department of Anaesthesiology, Coimbatore Medical College and Hospital, during the academic year 2013-2016.

Signature of the Guide
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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled **"A COMPARATIVE STUDY ABOUT THE EFFECT OF DEXMEDETOMIDINE VERSUS LIGNOCAINE ON HEMODYNAMIC AND RECOVERY RESPONSES DURING TRACHEAL EXTUBATION"** is a bonafide and genuine research work carried out by me under the guidance of ***Dr. T. SADAGOPAN., M.D., DA., Professor***, Department of Anesthesiology, Government Medical College and Hospital, Coimbatore, Tamil Nadu, India.

DATE:
PLACE: Coimbatore

Signature of the Candidate
Dr.J. LAKSHMI

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At the culmination of my study, I acknowledge my deep gratitude to all those patients who had volunteered themselves as subjects for this study.

DATE: **Signature of the Candidate**

PLACE: Coimbatore Dr. J. LAKSHMI

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INTRODUCTION

Extubation of the trachea is the process of discontinuing the artificial airway when the necessities for its use like ventilation, protection of the airway, obstruction of the airway and hypoxia were corrected. It is one of the most uncomfortable state during general anaesthesia. It is almost always associated with hemodynamic changes.[1]

Extubation during lighter planes of anaesthesia or sedation can stimulate reflexes by laryngeal and tracheal irritation. The laryngopharyngeal stimulation is associated with reflex increase in sympathetic activity leading to hemodynamic changes[2,3]. These hemodynamic changes are reflected as rise in heart rate and arterial blood pressure and are usually variable, transitory and unpredictable[1,4].

It is more dangerous in patients who have systemic hypertension, heart disease, intracranial aneurysms and cerebro vascular disease. Even the transient changes in arterial blood pressure and heart rate can result in potentially deleterious effects like cerebral hemorrhage, arrythmias, myocardial ischemia, left ventricular failure, pulmonary edema, and rupture of intracranial aneurysms[5,6].

For the endotracheal extubation to be smooth, the patient should not have any straining, coughing, bucking, movement, holding of breath, laryngo spasm or broncho spasm[7.]

Various drugs and techniques have been tried from time to time to attenuate the airway and stress responses during tracheal extubation. But none of the method has been completely successful.[8-13]

Trials have been conducted to attenuate the hemodynamic and stressor responses during tracheal extubation by using various drugs like opioids, inhalational agents, local anaesthetics, vasodilators, alpha blockers, beta blockers and calcium channel blockers.[12]

Studies have been carried out using fentanyl[8], sevoflurane, Lignocaine[9-11], propofol, magnesium sulphate, nitroglycerine, clonidine, esmolol[12], labetolol[13], metoprolol, verapamil, nicardipine, diltiazem[2,3], etc., either as a sole agent or in comparison with each other.

With this background, this study was conceptualized to analyze the outcome of dexmedetomidine with intravenous lignocaine on the hemodynamic and recovery profiles during endotracheal extubation.

AIM OF THE STUDY

The purpose of this study is to analyze and compare the properties of dexmedetomidine with that of lignocaine on the hemodynamic changes and variations in the recovery profile that occur during endotracheal extubation.

OBJECTIVES OF THE STUDY

1. To study the hemodynamic effects of dexmedetomidine and lignocaine on the patient during extubation.
2. To compare the quality of extubation of dexmedetomidine with that of lignocaine with respect to the patient's responses.
3. To study the emergence - agitation response of the patient with dexmedetomidine and lignocaine during and following the endotracheal extubation.

ANATOMY OF THE AIRWAY REFLEXES

ANATOMY OF THE AIRWAY REFLEXES

Anatomically, airway is the passage through which air passes during respiration. It is divided into an upper and a lower respiratory tract. Cricoid cartilage forms the boundary between the two.

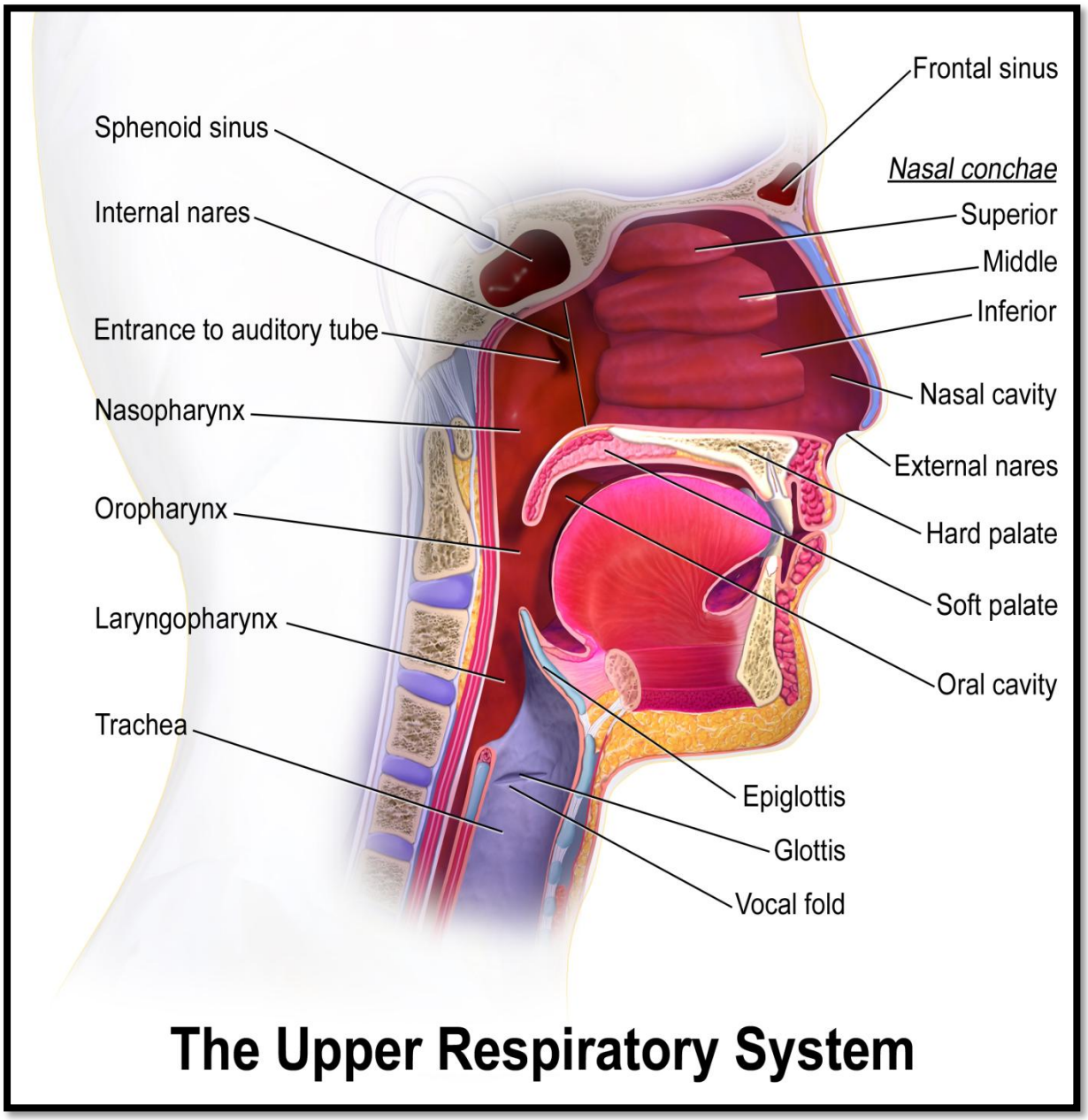


FIGURE : 1 UPPER RESPIRATORY SYSTEM

The upper airway consists of

i) External nostrils

ii) Nose

iii) Mouth

iv) Pharynx and

v) The larynx[14.]

NASAL CAVITY:

The nasal cavity is separated into two by the nasal septum. The floor of the nasal cavity is parallel to the hard palate.

The lateral wall consists of

i) Superior turbinate,

ii) Middle turbinate and

iii) Inferior turbinate.

The space below these constitutes the meatus[15.]

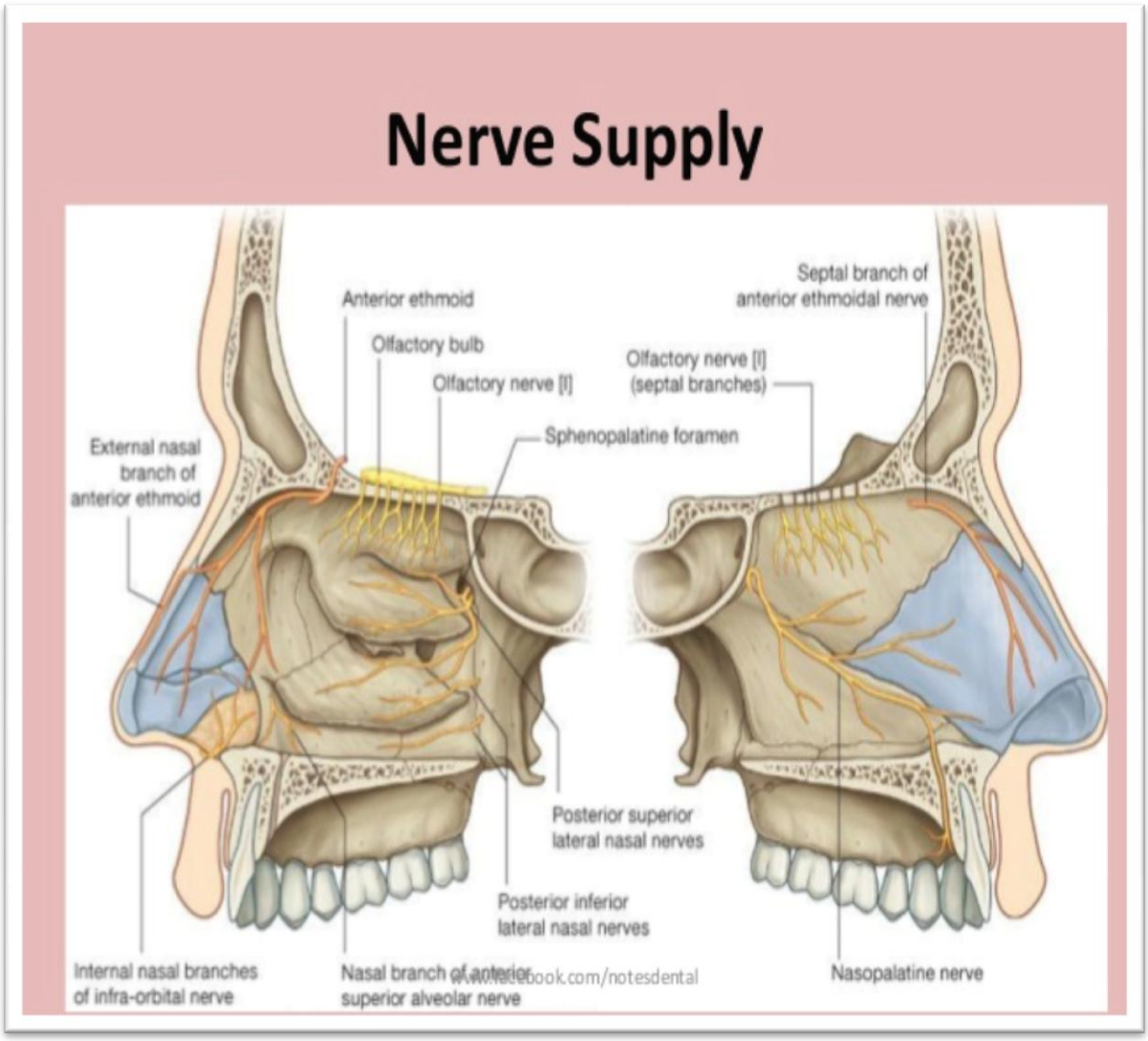


FIGURE 2 : NASAL CAVITY

The nerve supply to the nasal cavity is as follows:

i) Anterior ethmoidal nerves.

ii) Posterior ethmoidal nerves.

iii) Anterior - superior alveolar branch and

iv) Infra orbital branch of maxillary nerve.[15]

ORAL CAVITY:

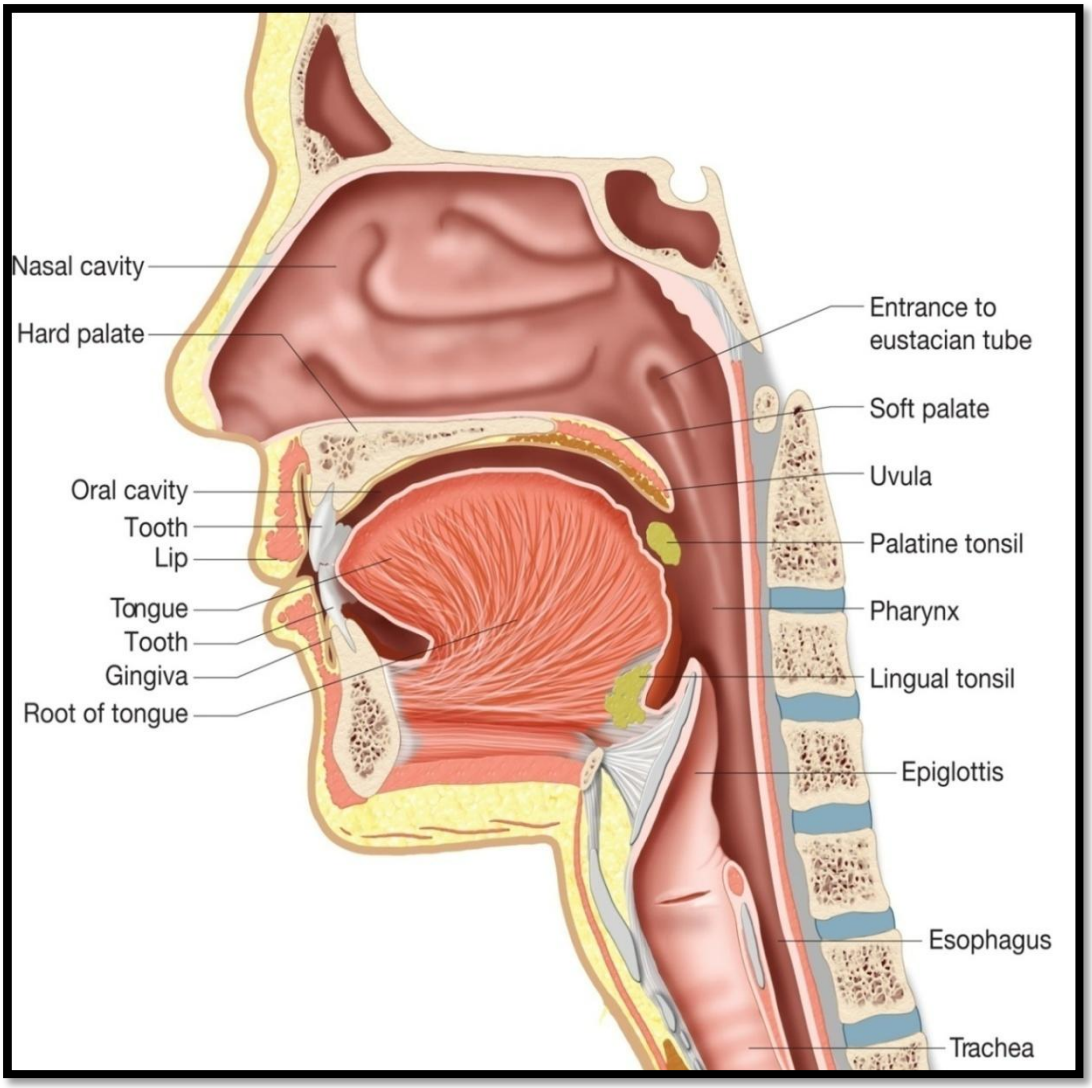


FIGURE 3 : ORAL CAVITY

The oral cavity consists of

i) Upper and lower dentition

ii) Tongue

iii) Floor of the mouth

iv) Hard palate

v) soft palate and

vi) The major salivary glands.

The nerve supply of the oral cavity is as follows:

i) Trigeminal nerve,

ii) Facial nerve,

iii) Glossopharyngeal nerve and

iv) Hypoglossal nerve.[4]

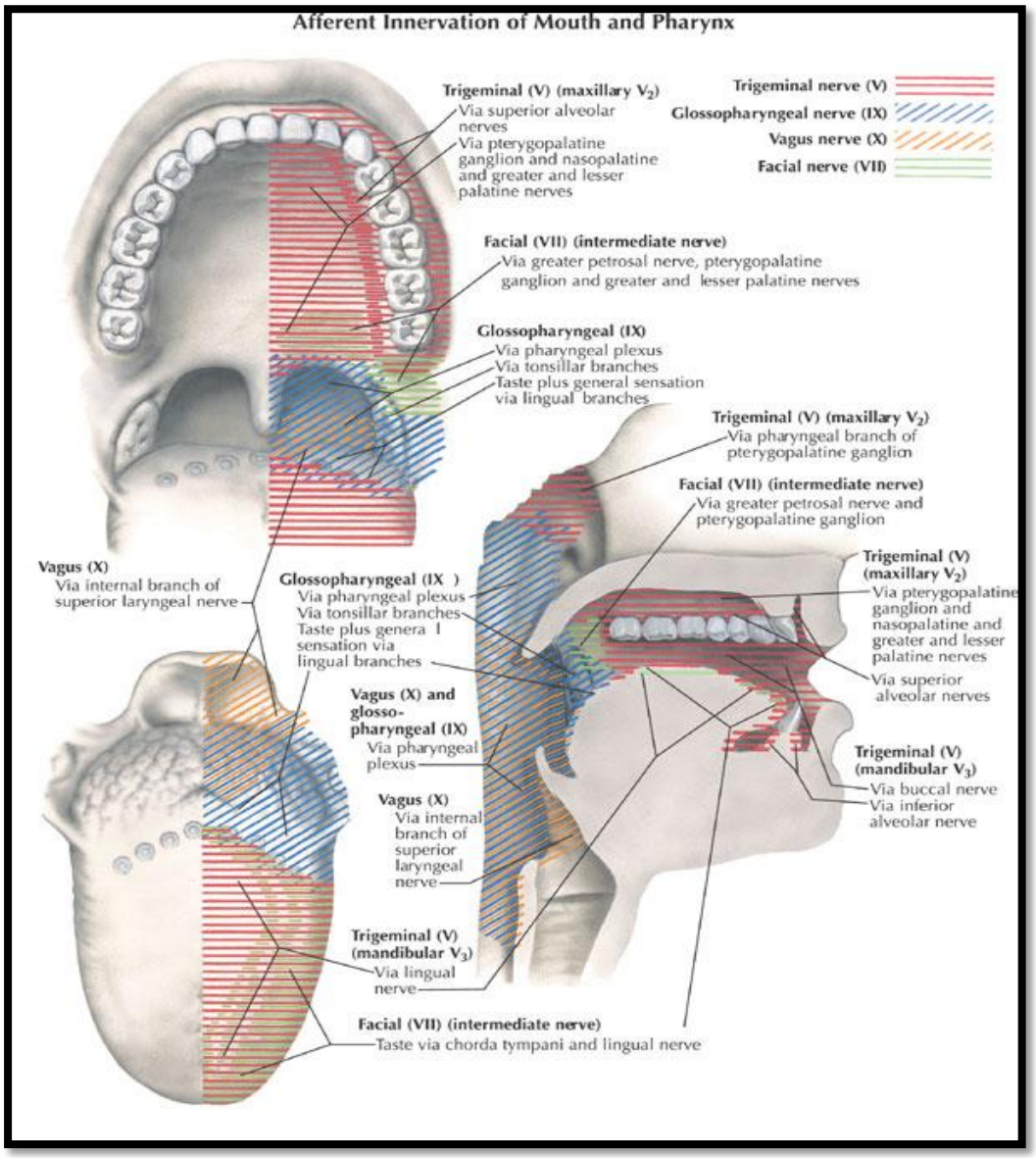


FIGURE 4: INNERVATION OF MOUTH AND PHARYNX

PHARYNX :

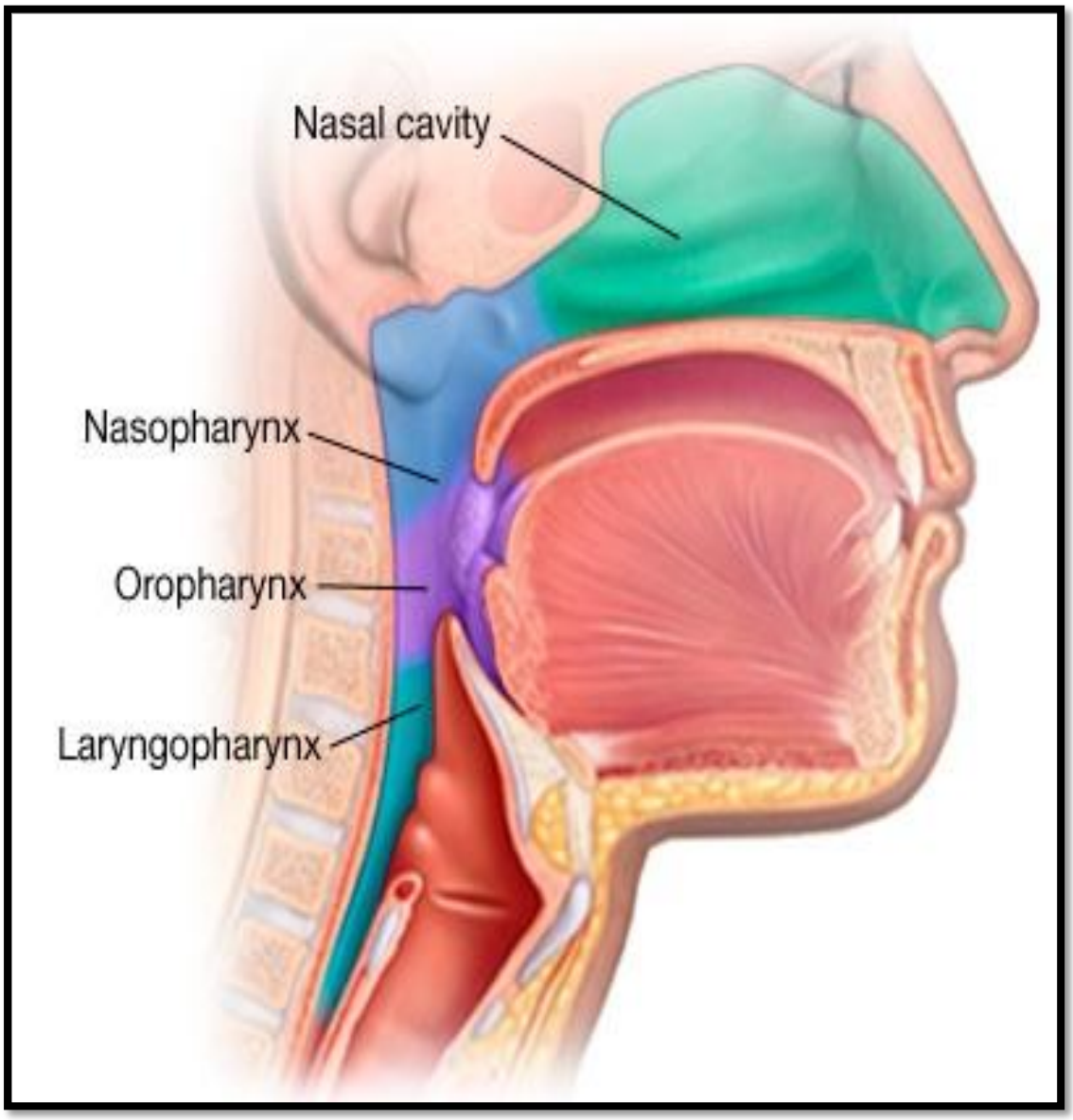


FIGURE 5 : PHARYNX

Pharnyx – 3 parts[16] :

i	Nasopharynx
ii	Oropharynx
iii	Laryngopharynx

The extensions[14] of these are as follows :

Nasopharyx	Above : from skull base. Below : soft palate.
Oropharynx	Above : soft palate. Below : hyoid bone - body.
Laryngopharynx	Above : hyoid bone - body Below : cricoid cartilage – lower border

NERVE SUPPLY:

Glossopharyngeal nerve supplies

i) Base of the tongue,

ii) Upper part of the epiglottis and

iii) The pharyngeal walls.

Superior laryngeal branch of the vagus nerve[18] supplies

i) Lower part of the epiglottis and

ii) The supra glottic parts.

LARYNX :

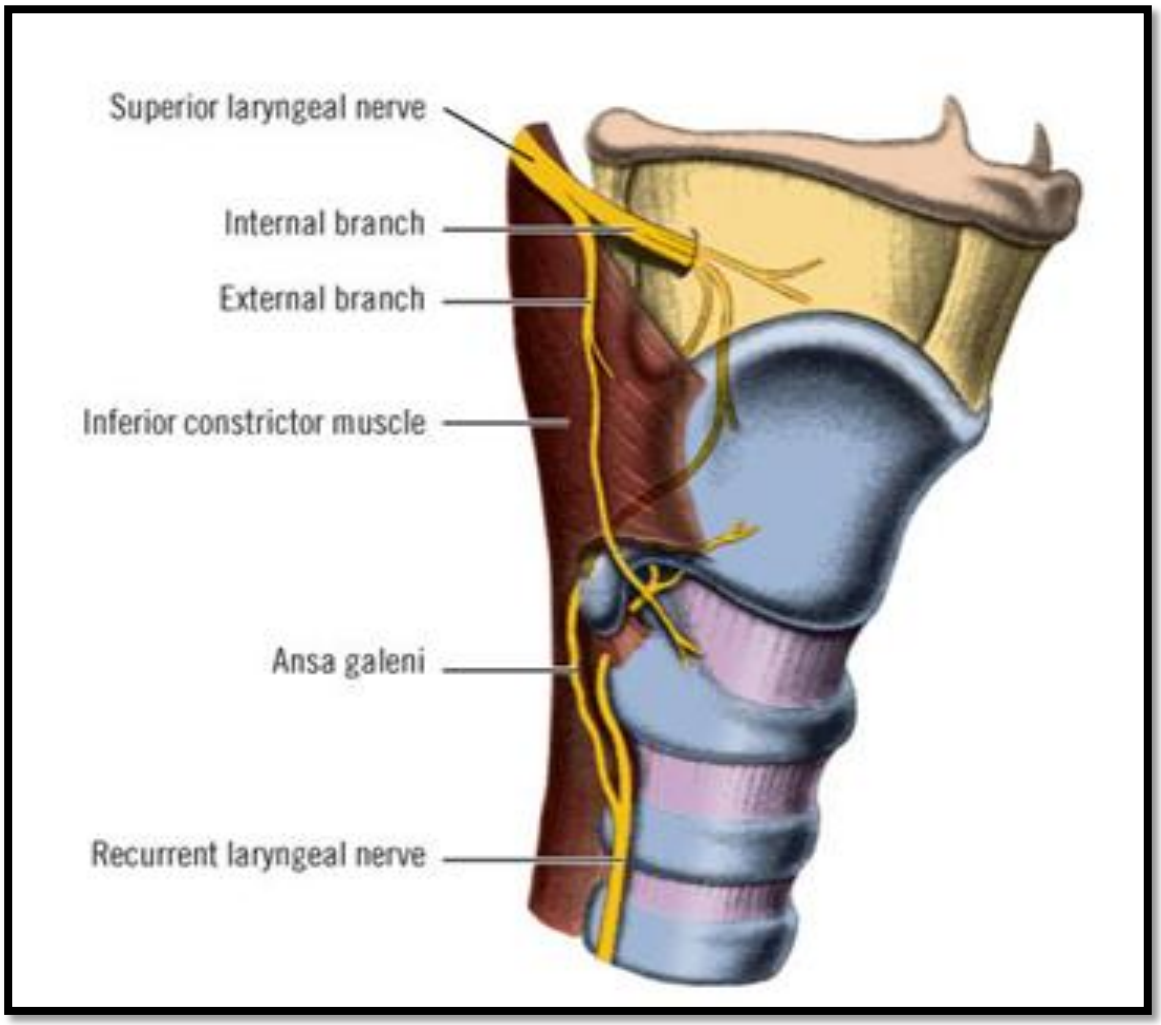


FIGURE : 6 LARYNX

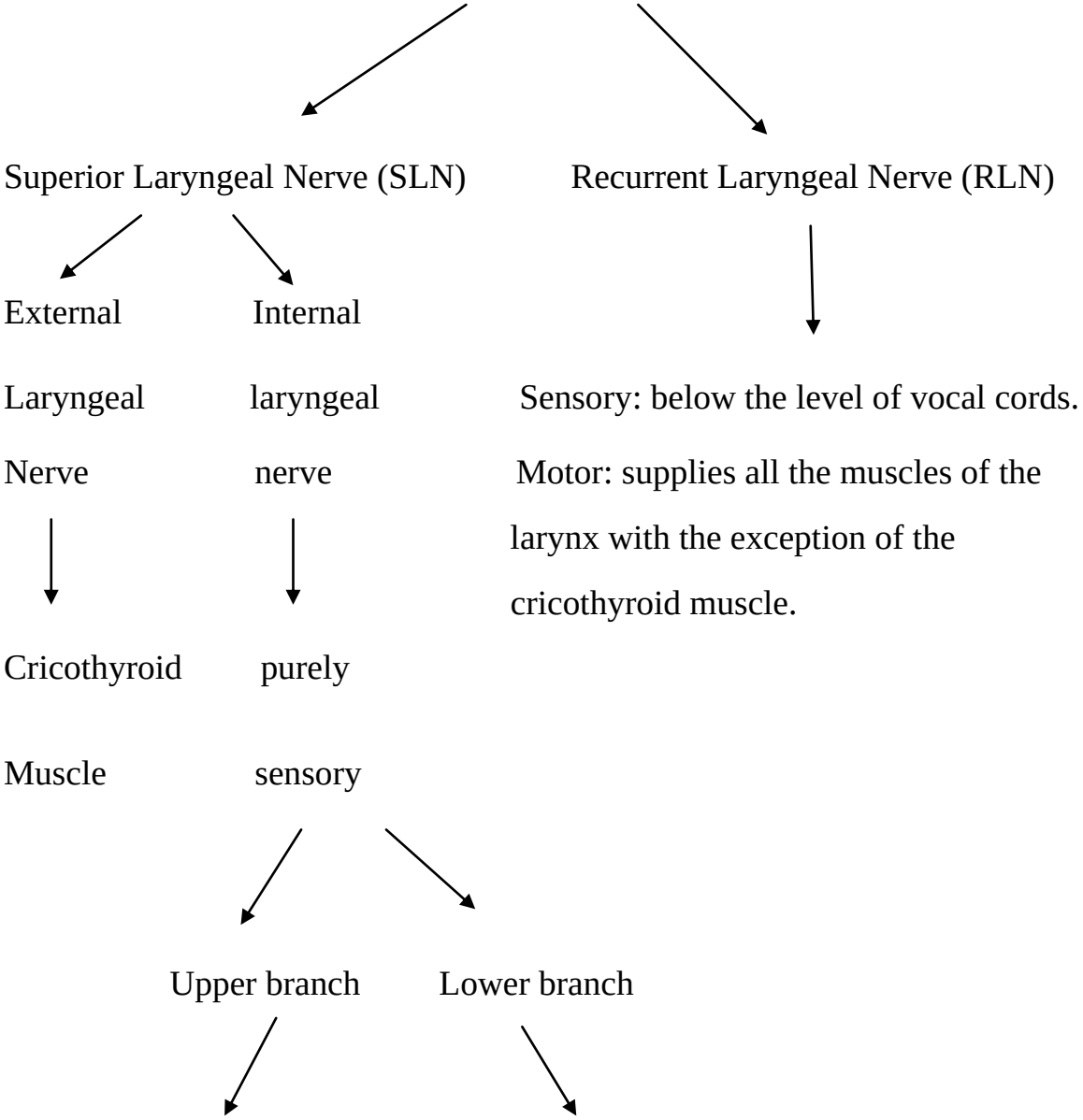
The larynx extends from C 3 – C 6 vertebrae. The structure consists of muscles, ligaments and a framework of cartilages[17].

The mucous membrane of the larynx receives its nerve supply from the

i) Superior Laryngeal Nerve (SLN)

ii) Recurrent Laryngeal Nerve (RLN).

NERVE SUPPLY OF LARYNX[15 - 18]



PHYSIOLOGY OF THE AIRWAY REFLEXES

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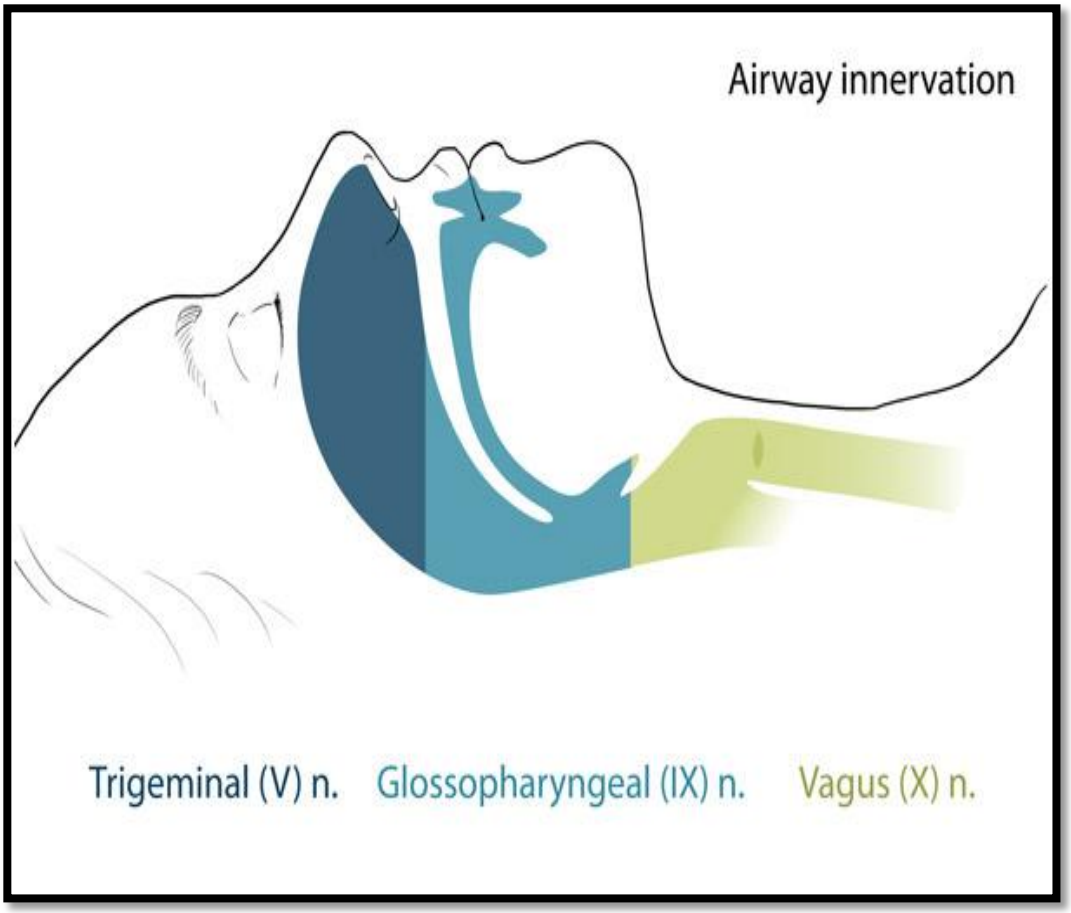


FIGURE 7 : AIRWAY INNERVATION

Lower part of the pharynx, epiglottic folds and the laryngeal wall has many sensory receptors that react to chemical, mechanical and thermal signals[4]. These mechanical receptors are densely distributed mainly in the epiglottic folds, vocal cords and lower pharyngeal wall. On stimulating these mechanoreceptors, motor reflexes like coughing, hiccup, pressor response and stimulation of the sympathetic system occurs[4,18].

These sensory receptors have many nerve endings which are present embedded in the epithelial tissues of the airway. They are found distributed densely on the laryngeal side of the epiglottis and over the arytenoids cartilages.[4] The afferent signals from the superior laryngeal nerve are carried by myelinated nerve fibres like A delta, B and C fibres. The recurrent laryngeal nerve has an abundant number of rapidly adapting sensory receptors which are mainly distributed on the vocal cords[4,18].

Afferent nerve fibres of the larynx project to the nucleus of tractus solitarius particularly in their posterior and caudal areas with medulla as the central projection site [4,14.]

Each efferent sympathetic pathway is composed of a pre-ganglionic neuron. The cell bodies of the pre - ganglionic neurons lie within the thoracic and upper lumbar spinal cord. These fibres pass from the spinal cord via anterior routes of each spinal nerve and then via the white ramus to synapse with post ganglionic cell bodies which are located within the ganglia of the sympathetic chains. From these ganglia, the post ganglionic sympathetic nerves pass to their effector organs. Pre ganglionic fibres of T8 to T12 synapse in the adrenal medulla[4,14]. Stimulation of these, causes release of catecholamines from the adrenal medulla into the circulation, thereby causing hemodynamic changes.[5,7]

Suggested mechanism of haemodynamic response :

TRACHEAL EXTUBATION

STIMULATION OF MECHANORECEPTORS IN LARYNX

AFFERENT STIMULI THROUGH IX AND X CRANIAL NERVES TO MEDULLA

REFLEX ACTIVATION OF VASOMOTOR CENTRE

SYMPATHETIC NEURAL OUTPUT

ADRENAL MEDULLA

HEART

RELEASE OF CATECHOLAMINES

BLOOD VESSELS

HEMODYNAMIC CHANGES

- TACHYCARDIA
- HYPERTENSION
- RAISED INTRACRANIAL PRESSURE
- RAISED INTRAOCULAR PRESSURE

DEXMEDETOMIDINE

DEXMEDETOMIDINE

FIGURE : 8 DEXMEDETOMIDINE

GENERAL INTRODUCTION

Dexmedetomidine is a highly specific agonist of the alpha – 2 receptor that has sympatholytic, analgesic, hypnotic, sedative, and anxiolytic properties. It was first licensed for the use of sedation in mechanically ventilated adult intensive care patients for up to 24 hours. [19]

In 2008 it was labelled for use as a sedative in non - intubated adult patients before surgical, diagnostic and therapeutic procedures.

Dexmedetomidine is not approved for use in any paediatric setting.

Despite these licensing restrictions, dexmedetomidine has been used extensively throughout the hospital setting for numerous off-label applications including,

1) Management of postoperative pain,
2) As an adjunct to anaesthesia in adult and paediatric patients[20],
3) Used in intensive care and procedural sedations for both adults and children,
4) Treatment of cyclic vomiting syndrome,
5) Treatment of shivering after anaesthesia,
6) Withdrawal / Detoxification amelioration in adult and paediatric patients[21]

PHYSICOCHEMICAL CHARACTERISTICS

Chemical description	(+) – 4 - (S) - [1 - (2, 3 – dimethylphenyl) ethyl] – 1 H - imidazole Monohydrochloride[21]
	S enantiomer of medetomidine
Subclass	Imidazole
Molecular weight	236.7
Solubility	Readily soluble in water
Empirical formula	C13H16N2.HCl

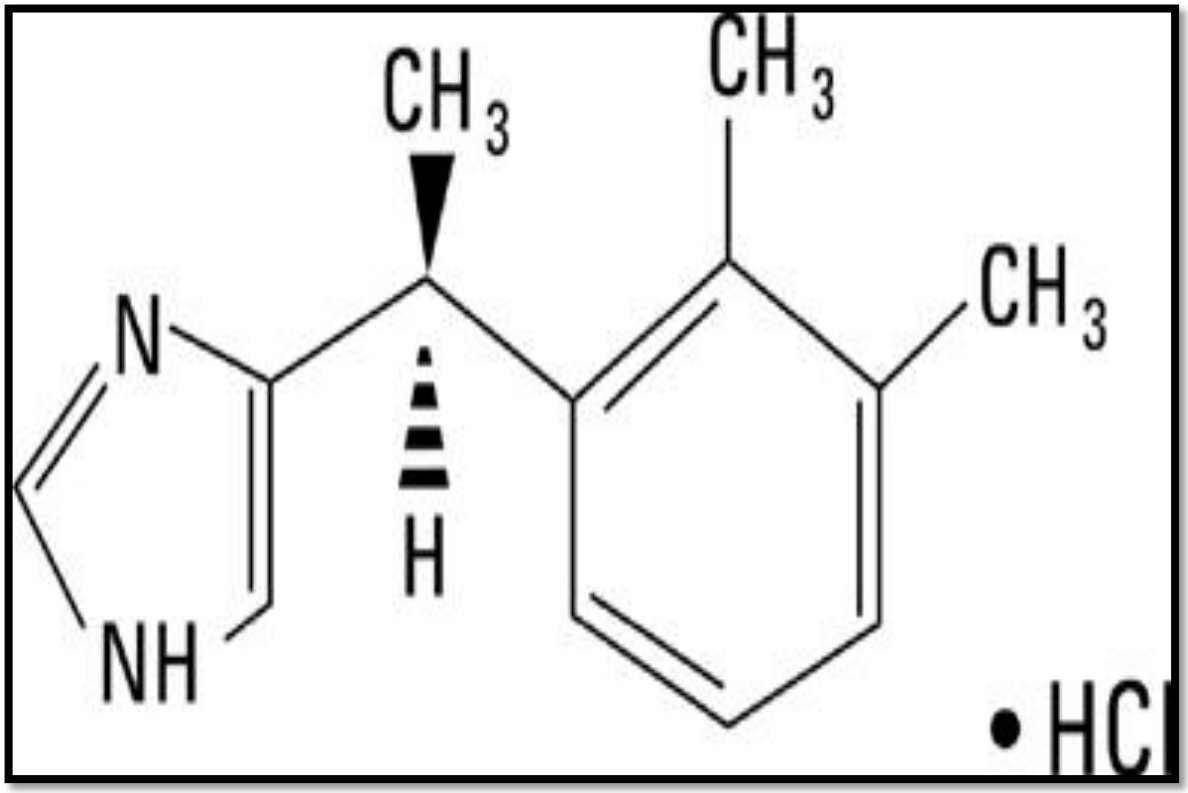


FIGURE 9 : CHEMICAL STRUCTURE OF DEXMEDETOMIDINE.

AVAILABILITY :

Dexmedetomidine is available in the form of 2-mL vials containing 100 μg /mL or 50 μg / ml or 50 μg / 0.5 ml solution. For adult patients :

Loading dose	1 mcg / kg given over a period of ten minutes
Maintainence dose	0.2 - 0.7 mcg / kg / hour

METABOLISM AND PHARMACOKINETICS

Distribution	Rapidly distributed
Protein binding	94 %
Metabolism	Liver
	Hydroxylation n-methylation (21%) conjugation (41%),
Excretion	Urine and faeces.

Having profound effects on cardiovascular variables, it may alter its own pharmacokinetics[22]. With large doses, there is marked vasoconstriction, which probably reduces the volume of distribution. In essence, Dexmedetomidine displays nonlinear pharmacokinetics. Dyck and collegues found that its pharmacokinetics in volunteers is best described by a three compartment model.[23]

Elimination	**Elimination Half-Life(hr)**	**Clearance (mL/kg/min)**	**VdSS (L / kg)**
Dexmedetomidine	2 - 3	10 - 30	2-3

These pharmacokinetic parameters apparently are unaltered by age, weight or renal failure, but clearance is a function of height. Postoperative patients sedated with Dexmedetomidine displayed pharmacokinetics similar to the pharmacokinetics seen in volunteers.

PHARMACOLOGY

Dexmedetomidine has a high specificity for the alpha 2 adrenergic receptor with the ratio between alpha 2: alpha 1 being 1600: 1. Clonidine which is also an alpha - 2 adrenergic receptor exhibits a ratio between alpha 2: alpha 1as 200: 1. Moreover, dexmedetomidine has a shorter half life of about two to three hours whereas clonidine has a long half life of about twelve to twenty four hours[24].

Dexmedetomidine exerts its physiologic actions through potentiation of the postsynaptically situated alpha - 2 adrenergic receptors which causes activation of G proteins leading onto a negative feedback effect and less release of adenylyl cyclase[21]. This causes a decrease in the activity of the cyclic adenosine monophosphate (cAMP) that is situated intracellularly following which the ion transmission channels get dephosphorylated. This contributes to the reduced activity of the nervous system leading onto the sedative and hypnotic effects of dexmedetomidine[21,22] .

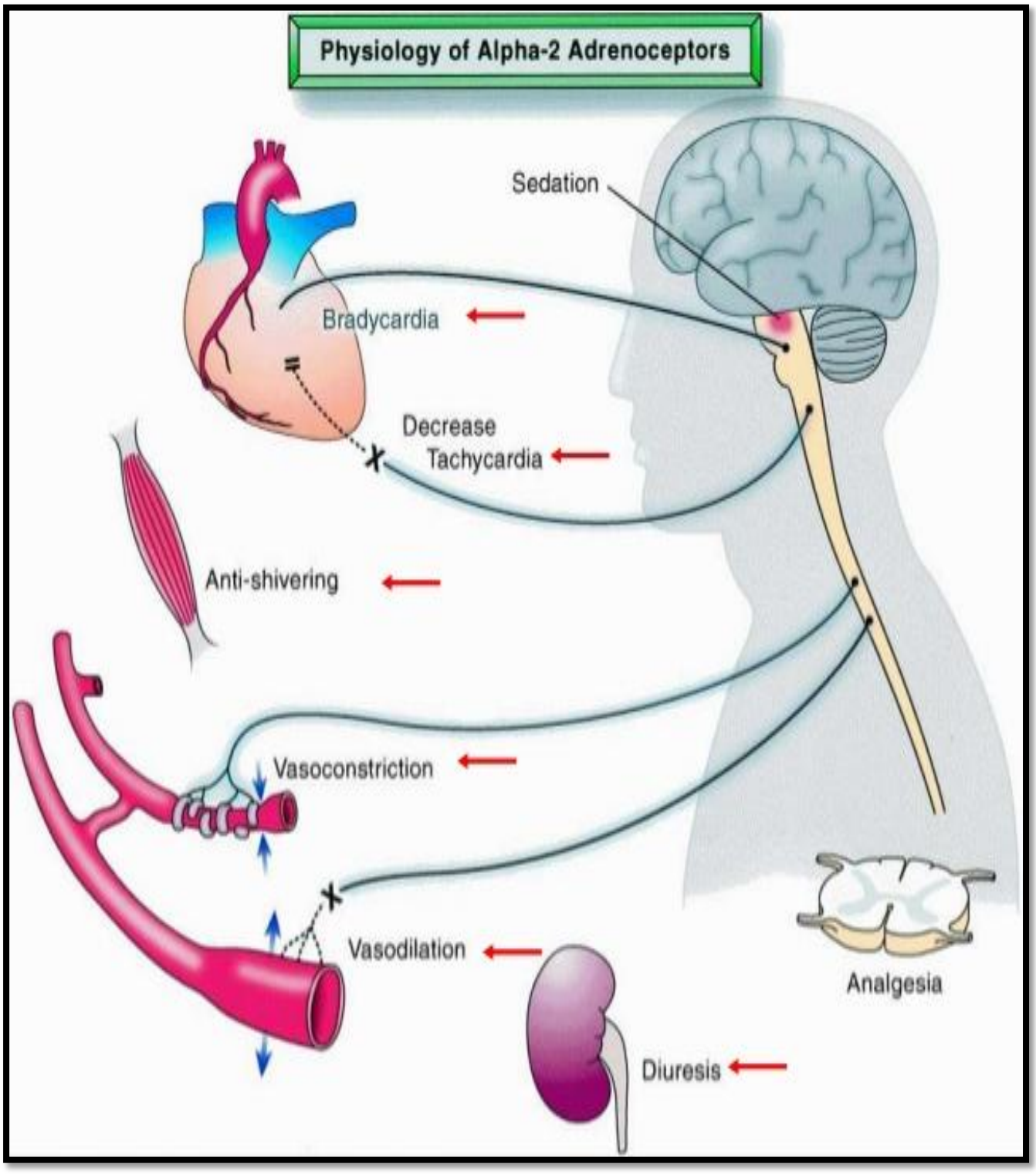


FIGURE 10 :PHYSIOLOGY OF ALPHA -2 ADRENOCEPTORS

The alpha – 2 adrenergic receptors are classified into three subtypes in human beings:

SUBTYPE	LOCATION
Alpha 2 A	Periphery
Alpha 2 B	Brain, spinal cord
Alpha 2 C	Brain, spinal cord

The postsynaptic alpha - 2 adrenoreceptors that are located in the peripheral blood vessels produce vasoconstriction, whereas the central action is through the activation of presynaptic alpha – 2 receptors in the medulla, which results in reduction of the norepinephrine turn over and suppression of the sympathetic discharge. Other actions include potentiation of the parasympathetic discharge and suppression of sympathetic discharge from the locus ceruleus. This action on locus ceruleus (LC) contributes to the sedative and anxiolytic properties of dexmedetomidine[26].

The stimulation of alpha – 2 adrenoreceptors which are situated in the posterior horn of the spinal tract causes inhibition of substance P, thereby exhibiting the analgesic properties of dexmedetomidine[27].

EFFECTS ON THE CENTRAL NERVOUS SYSTEM

SEDATION

Dexmedetomidine exerts the properties of anxiolysis and sedation by stimulation of the alpha - 2 adrenergic receptors which are situated in the locus ceruleus (LC) of the central nervous system.

The locus ceruleus plays a major role in a wide range of activities in the central nervous system, which includes somnolence, wakefulness, apprehension and syndromes that occur due to the withdrawal of drugs like central nervous system depressing agents and opiates .The sedation produced by these alpha - 2 adrenergic agonists are different from the sedation that is induced by drugs like propofol or benzodiazepines and does not seem to involve the cerebral cortex[28]. The sedative effect exhibited by dexmedetomidine is usually smooth and the patient responds to commands. The patient can be made to exhibit activities from somnolence to wakefulness and they can even perform work and they may become somnolent again once the stimulation is withdrawn.

The alpha 2 agonistic agents exert their action through specific pathways which are situated endogenously and stimulate sleep. This leads to a decrease in the activity of the projections of the locus ceruleus to the ventrolateral preoptic nucleus. This increases GABA and galanin release in

the tuberomammillary nucleus, producing a decrease in the histamine release in cortical and subcortical projections[29]. The sleep state that is produced by dexmedetomidine is similar to the natural sleep and hence, it is utilised in sedating ICU patients.

Similar to other adrenergic receptors, the alpha - 2 agonists also exhibit tolerance after prolonged administration. Dexmedetomidine can be employed for addiction treatment like cocaine withdrawal, rapid opioid detoxification, and iatrogenic induced benzodiazepine and opioid tolerance after prolonged sedation.

ANALGESIA

Dexmedetomidine produces analgesia by acting on the alpha - 2 receptors that are located in the central nervous system. The exact process by which dexmedetomidine produces analgesia is not yet studied completely. Brain tissue, spinal cord and other mechanisms seem to play a role in analgesia [30].

The main site which induces analgesia is found to be the spinal cord. Here, the stimulation of alpha - 2 C adrenergic receptor subtype is found to potentiate the analgesic effect of opioid drugs thereby decreasing the transmission of pain signals to brain centres[31].

Alpha - 2 agonistic agents do not exhibit analgesia when administered through the epidural or subarachnoid pathway. Clonidine injected in the neural axis helps with short term pain, cancer pain, and neuropathic pain. Systemic use of Dexmedetomidine also have demonstrated a narcotic sparing effect.

EFFECTS ON THE RESPIRATORY SYSTEM

Alpha - 2 agonistic agents exerts less influence on the respiratory system. It is found that dexmedetomidine when administered to the normal individuals or in the patients perioperatively, has not caused depression in the respiratory activity. It was observed that there is no variation between placebo and dexmedetomidine in the functions of respiratory system in the ICU patients[32]. Dexmedetomidine has not caused depression of the respiratory system even when administered in doses greater than the recommended levels[33].

EFFECTS ON THE CARDIOVASCULAR SYSTEM

The alpha – 2 B receptors are situated in the smooth muscle of the vascular wall and they cause constriction of the blood vessels[34].

In the central nervous system, activation of the alpha - 2 adrenergic receptors results in a decrease in the discharge of the sympathetic signals[26] and a rise in the parasympathetic activity. Dexmedetomidine also blocks the

ganglionic receptors that are situated in the periphery, and hence results in further accentuation of the parasympathetic activity. All these changes result in a predominant decrease in the catecholamine levels, and hence, a fall in the heart rate and a modest decrease in arterial blood pressure occurs[27].

Dexmedetomidine when administered as a bolus dose in human beings have exhibited a biphasic action. An acute intravenous administration of 2 mcg / kg has caused a transient rise in arterial blood pressure by 22% and a fall in heart rate by 27% from the baseline after about five minutes following intravenous injection. This transient rise in blood pressure is caused by the vasoconstricting property of dexmedetomidine due to the stimulation of the peripherally situated alpha 2 receptors[28]. Following a transient fall, the heart rate returns to its baseline value after about fifteen minutes, and the blood pressure slowly decreases and reaches a value that is less than 15 % from the baseline value at the end of one hour.

When dexmedetomidine is administered intramuscularly, transient rise in blood pressure did not occur and there was less than ten percent fall in blood pressure and heart rate. Bradycardia was also reported, mainly in young individuals who have a predominant vagal activity. It was found that beta blockers do not potentiate the effects of bradycardia[35].

Dexmedetomidine has been found to cause hypotension when administered to patients who are volume depleted. This can be minimized by

omission of the initial loading infusion and not administering a dose greater than 0.4 mcg / kg. The hypertension which occurred transiently during the initial period can also be minimized by administering the initial dose of drug slowly over a period of twenty minutes.

In several studies after intramuscular and intravenous administration, dexmedetomidine caused in a small percentage of patients, profound bradycardia (< 40 beats / min) and occasionally sinus arrest[36]. Generally, these episodes of bradycardia resolved spontaneously or were readily treated by anticholinergic agents without adverse outcome. It would be expected from its profile that dexmedetomidine would be beneficial to the ischemic myocardium. In animal models, dexmedetomidine showed some beneficial effects on the ischemic heart through decreased oxygen consumption and redistribution of coronary flow from non ischemic zones to ischemic zones after acute brief occlusion. No rebound effects have been found when discontinuing dexmedetomidine drips, even when it is given for more than 24 hours[37].

RENAL AND ENDOCRINE EFFECTS:

The alpha - 2 agonistic agents have a property of decreasing the neurohumoral stress responses[38]. It is observed that these agents when used for a period less than twenty four hours has not caused significant changes in the level of cortisol in the serum. In addition, dexmedetomidine has been

shown to suppress antidiuretic hormone and thereby causing natriuresis[39]. In resting volunteers, dexmedetomidine increased growth hormone secretion in a dose - dependent manner, but it had no effect on other pituitary hormones.

OTHER EFFECTS:

GASTROINTESTINAL SYSTEM:

During the intraoperative and postoperative period, the motility of the gastrointestinal system gets altered leading onto a delay in the emptying of the gastric contents. These factors have to be kept in mind while administering drugs for sedation and analgesia to the patients. It was observed that dexmedetomidine inhibits the emptying of gastric contents to a much lesser extent[40] when compared with that of morphine.

IMMUNE SYSTEM:

Studies have shown that many of the anaesthetic drugs that we use were found to inhibit the leukocytes. It is observed that dexmedetomidine does not alter the leukocyte function and hence can be used safely during acute inflammatory and infectious periods[41].

DEXMEDETOMIDINE CLINICAL APPLICATIONS

USES

Dexmedetomidine is found to have a wide range of clinical applications like sedation, anxiolysis, sympatholysis and analgesia with less depression of the respiratory system[26]. Hence, it is widely used in many clinical situations.

PREMEDICATION :

Many patients are prone for stress during the preoperative, intraoperative and postoperative period. Dexmedetomidine has been used successfully as an adjuvant to premedication because of its anxiolytic, sedative, analgesic, sympatholytic, and stable hemodynamic profile[26]. Dexmedetomidine also reduces the consumption of oxygen in the perioperative period. The premedication dose is 0.33 to 0.67 mg / kg intravenously given fifteen minutes before surgery and this dose minimizes the side effects of hypotension and bradycardia. In view of absent respiratory depression, it can be continued during extubation period unlike other drugs.

Dexmedetomidine potentiates the anaesthetic effect of all the anaesthetic agents irrespective of the mode of administration (intravenous, inhalation, regional block). Intraoperative administration of dexmedetomidine in lower concentrations has decreased the requirement of

other anaesthetic drugs[42]. But, side effects like bradycardia and hypotension are the limitations to its use necessitating the need for pharmacological rescue therapy. These effects can be accounted to the co – administered volatile anaesthetic agents which causes depression in myocardial contractility and vasodilatation. Dexmedetomidine when administered in high concentrations may cause increase in the systemic and pulmonary arterial pressure[43] due to the direct vascular changes that occur in the periphery and it may also cause a decrease in the contraction of the myocardium.

LOCOREGIONAL ANALGESIA

Dexmedetomidine is highly lipophilic in nature and hence, it readily crosses the brain blood barrier and is rapidly absorbed in the central nervous system. It binds to the alpha – 2 adrenoreceptors that are distributed in the spinal cord and exerts its analgesic action. It prolongs the duration of both sensory and motor blockade[45] caused by the local anaesthetic agents regardless of the way through which theses drugs are administered, for example, subarachnoid, caudal or epidural route. Dexmedetomidine enhances both the central and peripheral neural blockade caused by local anaesthetics; however, the peripheral neural blockade is due to its binding to alpha 2A – adrenoreceptor.

Dexmedetomidine has been successfully used in brachial plexus block, intravenous regional anesthesia (IVRA), and intra articularly.

0.5 mcg/ kg dexmedetomidine, when added to lignocaine has been found to improve the intensity and duration of the anaesthetic blockade. Addition of dexmedetomidine to levobupivacaine in brachial plexus blocks[46] has decreased the onset of action and enhanced the duration of the nerve blockade and provided better pain relief postoperatively. Intra articular administration of dexmedetomidine to patients undergoing arthroscopic knee surgery has been found to improve the intensity and quality of pain relief perioperatively[47].

For ICU sedation, dexmedetomidine has become a popular sedative agent because of its ability to produce cooperative sedation, i.e., patients remain awake, calm, and are able to communicate their needs. It does not interfere with the respiratory drive or produce any agitation, hence facilitating early weaning from the ventilator, thereby reducing overall ICU stay costs. Since dexmedetomidine maintains a sleep state that is similar to that of the natural sleep, it hastens the recovery in ICU patients. Dexmedetomidine, on comparison with the conventional opiates and sedatives, is found to have better analgesic and sedative properties, produces less delirium and has caused less depression of the respiratory system and also has a favourable outcome in the cardiovascular system.

PROCEDURAL SEDATION

Dexmedetomidine is an attractive agent for short term procedural sedation and has been safely used in elective awake fiberoptic intubation,

transesophageal echocardiography, awake carotid endarterectomy, colonoscopy, vitreoretinal surgery, shockwave lithotripsy, pediatric MRI[48] and in paediatric patients undergoing tonsillectomy. The loading dose of dexmedetomidine for procedural sedation is 1 mcg / kg and the dose for maintainence is 0.2 mcg / kg / hour. The onset time for its action is about five minutes and reaches the peak in about fifteen minutes. Atipamezole is the antagonist of choice for the alpha - 2 adrenoreceptor agents and hence it reverses the pharmacologic actions of dexmedetomidine.

CONTROLLED HYPOTENSION

Dexmedetomidine is a safe and an effective agent for controlled hypotension which is mediated by its central and peripheral sympatholytic action. Spinal fusion surgery for idiopathic scoliosis, tympanoplasty and septoplasty[49] operations and maxillofacial surgery have been safely done with dexmedetomidine -controlled hypotension.

Dexmedetomidine has a significant opioid sparing effect and is useful in intractable neuropathic pain. In cardiac surgeries, dexmedetomidine apart from attenuating the stress responses during intubation of trachea, has also contributed to a decrease in the area of myocardial ischemia.

NEUROSURGERY:

Dexmedetomidine provides a consistent cerebral hemodynamic effect without causing a sudden rise in intracranial tension during endotracheal intubation, extubation, and insertion of the head pin. It attenuates the

neurocognitive impairment (delirium and agitation) thereby, allowing immediate postoperative neurological evaluation[50]. Dexmedetomidine provides neuroprotection through various methods of action which makes it, a promising drug in neurosurgery. It does not interfere with neurological monitors and has an upcoming role in "functional" neurosurgical procedures like awake craniotomy which involves resecting the tumours of the brain and also in the surgical management of Parkinson's disease.

OBESITY

Dexmedetomidine does not cause respiratory depression and has been infused at the rate of 0.7 mcg/ kg intraoperatively to avoid respiratory depression due to narcotic usage in morbidly obese patients.

OBSTETRICS

Dexmedetomidine is being used effectively as an adjunctive agent in parturients who are in labour and in whom epidural analgesia was not effective. It provides anxiolysis, stable hemodynamics and also stimulates the contractility of the uterus. It is retained in placental tissue and passes less readily into the fetal circulation than clonidine because of its high lipophilicity and thereby has less susceptibility to cause fetal bradycardia.

PEDIATRICS

Dexmedetomidine is not approved yet for use in the paediatric population. Yet, it has been used successfully in inducing somnolence in the

paediatric patients in the ICU[51] and also for diagnostic and therapeutic radiological imaging techniques[48].

OTHER USES:

- Dexmedetomidine is found to be effective in the management of withdrawal symptoms associated with alcohol[52], opioids, benzodiazepines and other addicting drugs.
- As an adjunct in otorhinolaryngology anesthesia for rhinoplasty and in surgeries involving the middle ear.
- As an adjunctive agent in aneurysmal repair surgeries.
- As an antishivering agent.
- Dexmedetomidine is effective in preventing ethanol induced neurodegeneration.

DRUG INTERACTIONS

Dexmedetomidine when administered with other anaesthetic agents or sedatives is found to exhibit a drug interaction which results in the potentiation of the overall sedative effect. This adjunct effect often decreases the reliance on other agents. Concurrent administration of dexmedetomidine and digoxin may result in a decrease in the heart rate, possibly through an additive increase in vagal tone.

ADVERSE EFFECTS

Dexmedetomidine is associated with many adverse effects. This includes bradycardia, hypertension, hypotension, dry mouth, nausea, vomiting, atrial fibrillation, pyrexia, chills, pleural effusion, atelectasis, pulmonary edema, hyperglycemia, hypocalcemia, acidosis, etc. Rapid administration of dexmedetomidine infusion at the rate of 1 mcg / kg / hour if administerd in less than ten minutes may cause transient hypertension mediated by peripheral alpha 2B - adrenoreceptor vasoconstriction. But hypotension and bradycardia[53] may occur with ongoing therapy which is mediated by central alpha 2A – adrenoreceptor thereby causing decreased release of noradrenaline from the sympathetic nervous system. Long term use of dexmedetomidine leads to supersensitization and upregulation of the receptors; so, with abrupt discontinuation, a withdrawal syndrome of nervousness, agitation, headaches, and hypertensive crisis can occur.

LIGNOCAINE

LIGNOCAINE

FIGURE 11 :LIGNOCAINE

Lignocaine is a synthetic amide – linked anaesthetic agent of intermediate potency and duration of action. In 1943, Lofgren synthesized lignocaine first in Sweden. It was first used by Gordh in 1948[54].

Lignocaine is set as the standard drug to which other local anaesthetic agents are correlated. At present, it is the most commonly used local anaesthetic agent.

MECHANISM OF ACTION :

Lignocaine exerts its action by binding reversibly to the intracellular portion of the voltage gated sodium channels, thereby inhibiting the movement of sodium ions across the channel resulting in conduction blockade. This results in the slowing of depolarization, so that the threshold level is not achieved and hence, there is no propogation of action potential. But there is no change in the resting membrane potential. Lignocaine binds to the inner portion of the receptor (i.e., sodium channel) after entering the cell membrane[54,55].

PHYSICOCHEMICAL PROPERTIES :

It has a molecular weight of 234. It is a weak base with a pka of 7.6 – 7.8. It is very stable and is not decomposed by boiling, acids and alkalies.

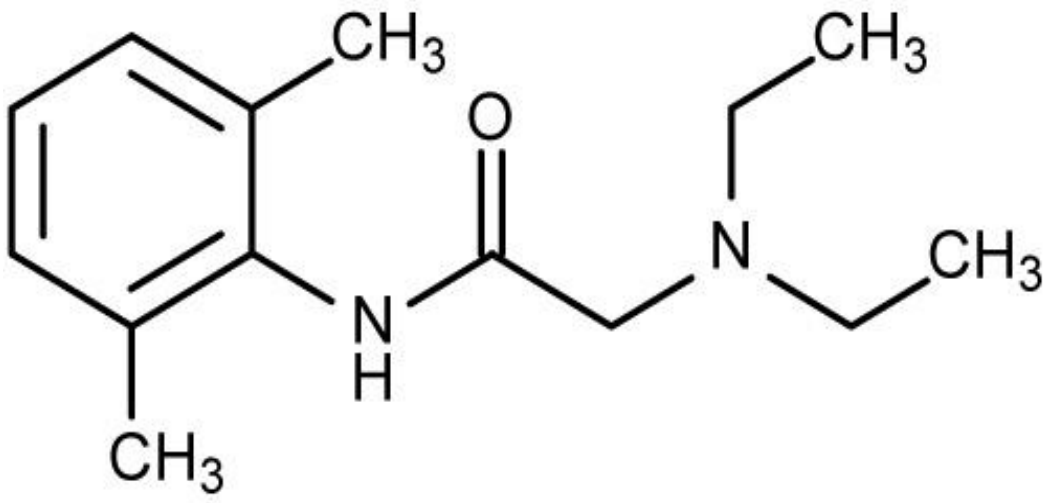


FIGURE 12 : CHEMICAL STRUCTURE OF LIGNOCAINE

PHARMACOKINETICS :

It is absorbed from the site of application or injection into the blood stream.

METABOLISM AND EXCRETION :

Lignocaine undergoes rapid metabolism in the liver. The metabolic end products that are formed are excreted unchanged by the kidney. The process of biotransformation[54] involves :

i) Oxidative N – dealkylation

ii) Hydroxylation of the ring

iii) Cleavage of the amide bondage

iv) Conjugation.

Oxidative N – dealkylation[56] which is the main step involved in biotransformation results in the formation of two metabolites :

i) Glycinexylidide

ii) Monoethylglycinexylidide.

The toxicological and pharmacological properties exerted by these metabolites shows similarity with that of lignocaine but has less potency. Nearly, 90 % of the lignocaine that is injected is excreted as metabolites and about a level of ten percent is excreted unchanged.

When lignocaine is administered intravenously as a bolus dose, it has an elimination half life of 90 to120 minutes[54]. Since, the metabolism of lignocaine is rapid, factors which causes alterations in the liver function can also cause changes in the pharmacokinetics of lignocaine. There may be prolongation of half life in patients who have derangements in their liver functions. The metabolism of lignocaine is not affected by the functioning of renal system but may result in the accumulation of its metabolites[56].

The systemic effects produced by lignocaine are affected by various parameters like a decrease in PH and usage of central nervous system stimulating and depressing drugs. The effects are more prominent, when the plasma level of the lignocaine in the venous blood rises more than 6 mcg / mL[54].

ONSET OF ACTION:

It has a rapid onset of action occurring for topical anaesthesia in 5-10 minutes. For conduction anaesthesia in small nerves, it is 5 - 10 minutes and for large nerves it is 10 - 15 minutes and for intravenous administration, it is 45 to 90 seconds[56]. It has a protein binding of 70% and is bound to α 1 acid glycoproteins with the volume of distribution being 91 litres.

Lignocaine has a triphasic distribution.

Rapid distribution phase (α): In this phase, the drug is distributed to highly vascular regions with t½ of 1 minute.

Next is slow disappearance phase (β): Here the drug is distributed to slowly equilibrating tissues with t½ of 9.6 min.

Last is slow transformation and excretion phase (δ): Where the t ½ is 1.6 hrs with clearance of 0.95 litres per minute[54].

AVAILABILITY:

a) 5% heavy 2 ml ampoules which contain 50 mg of lignocaine / ml with 75 mg – 100 mg of dextrose
b) 2% ligcocaine (xylocard) without preservative – 50 ml vial for intravenous use
c) 2% lignocaine – plain – 30 ml vial – containing methyl and propyl paraben as preservative.
d) 2% lignocaine with 1 in 200000 Adrenaline
e) 4% lignocaine with 1 in 200000 Adrenaline – 30 ml vial.
f) 4% lignocaine viscus
g) 4% lignocaine aqueous solution
h) 10% lignocaine spray
i) 2% lignocaine Jelly

j) 2% lignocaine ointment

k) 5% lignocaine ointment

PHARMACODYNAMICS:

LOCAL ACTIONS:

Lignocaine causes nerve blockade with loss of pain and temperature sensation, touch, motor power and vasomotor tone in the region supplied by the nerves.

SYSTEMIC ACTIONS:

This results due to the systemic absorption of the drug from the site of administration or through intravenous administration

1. CARDIOVASCULAR SYSTEM:

It has a stabilizing effect on the cell membranes of cardiac tissue. Lignocaine decreases the automaticity of the myocardium by opposing the depolarization of the phase IV of action potential spontaneously and thereby decreasing the effective refractory period. Lignocaine when administered in larger doses may cause decrease in the contractility of the myocardium and also a decrease in the velocity of conduction of impulses. These changes are caused due to the direct effects that occur in the membrane of the cardiac muscle which occurs as a result of blockade of the sodium channels[57].

It stabilizes the membrane of damaged and excitable cells, tending to suppress the ectopic foci.

2. VASCULAR SMOOTH MUSCLE :

In the vascular smooth muscles, lignocaine produces vasodilatation.

3. RESPIRATORY SYSTEM :

Whenever there is low arterial oxygen tension[54] (PaO2), lignocaine decreases the ventilatory response to hypoxia. Apnea may occur

i) Due to the paralysis of intercostal and phrenic nerves following direct contact with the local anaesthetics.

ii) Following suppression of the respiratory centre that is situated in the medulla.

Lignocaine also causes relaxation of the smooth muscles in the bronchi.

4. CENTRAL NERVOUS SYSTEM :

In the central nervous system (CNS), lignocaine produces a sequence of stimulation followed by depression. It produces sedation on intravenous administration. When lignocaine is administered intravenously, there is a decrease in the blood flow to the brain, particularly to the cerebrum, thereby resulting in a reduction in the rate of increase in the intracranial tension[54] which occurs during intubation and extubation. Infusion of lignocaine is

capable of reducing the Minimal Alveolar Concentration (MAC)[58] of volatile anaesthetics by 40%.

5. MUSCULOSKELETAL SYSTEM:

Lignocaine is myotoxic leading to lytic degeneration, edema and necrosis.

6. HAEMATOLOGICAL:

It decreases coagulation and enhances fibrinolysis.

INDICATIONS[54]:

1. For infiltration block, spinal, epidural, peripheral nerve blocks, topical anaesthesia and intravenous regional anaesthesia.
2. Antiarrythmic agent: Lignocaine is a class IB antiarrythmic agent. It is used to treat
   a) Ventricular tachyarrythmias.
   b) Arrythmias following acute Mycardial Infarction during cardiac surgery.
   c) In digitalis toxicity – because it does not worsen the trioventricular – block.
3. Prevention or treatment of increases in intracranial pressure during intubation - antitussive effect may be the reason.
4. Reflex induced bronchospasm is also attenuated by intravenous administration of lignocaine.

5. It suppresses noxious reflexes such as coughing and sympathetic stimulations associated with endotracheal suctioning and intubation[58].
6. It is used intravenously as an analgesic for certain chronic pain states.
7. It is used as a supplement to general anaethesia.

CONTRAINDICATIONS[55]:

1. Hypersensitivity.
2. Should not be used with vasoconstrictor in digits of hand, feet and penis.
3. It should not be given to patients with stokes adam syndrome and also in those patients who have various degrees of heart blocks involving the conducting system.

DOSES:

Maximum recommended dose[54] :

a) Plain - 3 mg / kg

b) With adrenaline - 7 mg / kg

c) For reflex suppression - 1.5 mg / kg iv.

DRUG INTERACTIONS:

1. Beta blockers: Coadministration of beta blockers, increases the serum levels of lignocaine and its toxicity, by decreasing lignocaine's metabolism.
2. Anticonvulsant agents: Increases lignocaine's metabolism.
3. Non depolarizing muscle relaxant blockade is potentiated by lignocaine.
4. Opioids and α2 adrenergic agonists : Potentiate lignocaine's pain relief.
5. Cimetidine : cimetidine decreases the blood flow to the liver and hence decreases the systemic clearance of the drugs which have a high extraction ratio. Studies have demonstrated that lignocaine when administered along with cimetidine has decreased the clearance of the drug from the body. It also caused an increase in the concentration of the drug in the serum by about fifty percent. Lignocaine when administered within the therapeutic range may result in increased toxicity of the drug when it is administered along with cimetidine.
6. Antiarrythmic agents : Potentiate the cardiac effects of lignocaine.

TOXICITY[59]:

Toxicity occurs mostly due to systemic absorption of locally administered lignocaine or due to accidental intravenous administration of large doses of lignocaine. The central nervous system is mostly vulnerable.

BLOOD LEVELS AND SYMPTOMS:

4 micrograms / ml : Light headedness, tinnitus, circumoral and tongue numbness (anticonvulsant and antiarrythmic activity)

6 micrograms / ml : visual disturbances

8 micrograms / ml : muscular twitching

10 micrograms / ml : convulsions

12 micrograms / ml : Unconsciousness

15 micrograms / ml : Coma

20 micrograms / ml : respiratory arrest

26 micrograms / ml : cardiovascular collapse

ADVERSE EFFECTS:

1. Allergic and hypersensitivity reactions: Due to the preservative used – methyparaben.
2. Cardiovascular system : Bradycardia, hypotension.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Barkha Bindu et al[60]., conducted a study in which they compared the hemodynamic and recovery profiles between dexmedetomidine and placebo at the time of endotracheal extubation. 50 patients were categorized into two categories – dexmedetomidine and placebo. They observed that dexmedetomidine stabilizes hemodynamics and facilitates smooth extubation.

Turan G et al[61], carried out a study in which they compared dexmedetomidine 0.5 ug / kg vs. 20 ml of 0.9 % NaCl for intracranial neurosurgery patients. They compared the systolic, diastolic and mean blood pressures and heart rates, the time taken for extubation and the extubation quality were analyzed. It was found that dexmedetomidine given intravenously five minutes before the completion of surgery provided stable hemodynamics, made extubation easier, and also provided better recovery and early neurological testing after neuro surgeries.

Tanskanen et al[62]., observed that a reduction in the rise in blood pressure during extubation was associated with the dose of the dexmedetomidine which was infused intravenously and it was found that a higher dose of dexmedetomidine was found to be more efficient in controlling blood pressure than a lower dose in patients who underwent removal of supratentorial tumours.

Aksu R et al[63]., carried out a double blinded randomized control study in 40 patients who underwent rhinoplasty with 20 patients in each category. Dexmedetomidine given at a dose of 0.5 mcg / kg before endotracheal extubation had a better profile when compared to that of fentanyl when given at a dose of 1 mcg in diminishing the stressor responses to endotracheal extubation and it also maintained stable hemodynamics without delaying the recovery.

Patel Cr et al[64]., compared the effects of dexmedetomidine with fentanyl on the intraoperative and postoperative hemodynamic profiles, entropy (response entropy and state entropy), postoperative sedation and recovery. It was found that dexmedetomidine has decreased the stress response at the time of endotracheal intubation and surgery to a greater extent and also maintained the hemodynamic stability.

Arain et al[65]., studied 34 patients who were posted for elective surgeries. First group of patients were administered dexmedetomidine 1 mcg / kg over a time of ten minutes and then 0.4 mcg / kg for four hours. Another group of patients received morphine sulphate at the dose of 0.08 mg / kg thirty minutes before the completion of surgery. They concluded that dexmedetomidine significantly reduced the opioid requirements by 66% in the postoperative period.

Talke P et al[66]., conducted a study in patients who underwent vascular surgeries in which they observed that dexmedetomidine decreased the rise in heart rate and norepinephrine levels in the plasma during recovery from anaesthesia and it did not decrease the rise in heart rate and blood pressure after recovery from anaesthesia. They also observed that dexmedetomidine did not affect the anaesthetic and analgesic requirements in the perioperative period.

Guler G et al[67]., observed that dexmedetomidine when administered five minutes prior to the completion of surgery intravenously at the rate of 0.5 mcg / kg over a period of sixty seconds has diminished the circulatory and airway reflexes at the time of tracheal extubation.

Peng K, Wu S, Liu H, Ji F[68] compared the effect of dexmedetomidine with placebo as an anaesthetic adjuvant for intracranial procedures. It was found that dexmedetomidine had a better hemodynamic profile perioperatively, less consumption of opiod drugs intraoperatively, and also had a lesser need for antiemetic drugs postoperatively.

Sanikop C, Bhat S[69]., conducted a study on the patients who underwent surgery for cleft palate repair. They administered intravenous lignocaine vs. placebo two minutes prior to tracheal extubation and studied its effect on the prevention of laryngospasm after extubation. It was observed

that lignocaine administered intravenously 1.5 mg / kg has prevented the laryngospasm following tracheal extubation.

Zamora Lozano J et al[70]., compared the effect of intravenous , topical and intracuff lignocaine in decreasing cough following extubation at the time of recovery. It was observed that intravenous lignocaine and intracuff lignocaine significantly decreased the cough response during emergence from anaesthesia.

Venkatesan T, Korula G[71] compared the effects of intracuff lignocaine and intravenous lignocaine which is administered at the dose of 1.5 mg / kg. They studied the cough response and hemodynamic profile at the time of endotracheal extubation in patients who underwent neurosurgery. They observed that the efficacy of the intracuff lignocaine was not greater than intravenous lignocaine in diminishing the cough response and hemodynamic profiles at the time of endotracheal extubation.

George SE et al[72]., conducted a study in which they compared intravenous lignocaine and the lignocaine which was administered through the endotracheal tube in patients who underwent craniotomy with skull pins. It was found that intravenous lignocaine has not prevented the cough response during tracheal extubation, if it was administered twenty to thirty minutes prior to tracheal extubation.

Sridhar P et al[73]., conducted a study in 134 patients with 67 patients in each group. One group of patients received an intravenous infusion of lignocaine 1.5 mg / kg at the time of intubation and then 1.5 mg / kg / hour throughout the intraoperative period and upto one hour after surgery. It was observed that intravenous lignocaine that was administered perioperatively has decreased the stress response to surgery, has provided better analgesia and has reduced the requirement of opioids in the post operative period.

Jain S et al[74]., conducted a study on the perioperative administration of intravenous lignocaine as infusion in sixty patients who underwent laparoscopic cholecystectomy. They concluded that intravenous administration of lignocaine has decreased the rise in mean arterial pressure and pulse rate during the perioperative period.

Lee JH et al[75]., conducted a study in seventy female patients who underwent thyroid surgeries. They studied the effect of intravenous lignocaine with that of remifentanil on recovery during anaesthesia and concluded that remifentanil decreased the stress responses to the endotracheal tube during recovery better than that of intravenous lignocaine.

SUBJECT AND PURPOSE OF STUDY

Tracheal intubation and extubation are among the most stressful events to the patient during general anaesthesia which are associated with acute sympathetic stimulation and hemodynamic changes. The stress response to intubation can usually be controlled by the effect of induction agents and opioids itself. Extubation is a different scenario where all the drugs are withdrawn from the patient and so there is always an exaggerated stress response leading onto severe hemodynamic changes. For an uncomplicated tracheal extubation, the patient should not cough, buck, hold breath or strain on the endotracheal tube.

Dexmedetomidine has a high selectivity for the alpha - 2 receptor and it also has a high potency. Dexmedetomidine, as an agonist at the alpha – 2 receptor exhibits the following properties :

i) Sympatholysis

ii) Sedation

iii) Sparing of other anaesthetic drugs

iv) Providing stable hemodynamic characteristics without causing significant respiratory depression.

Lignocaine is an amide synthetic local anaesthetic with antiarrythmic properties. It has been used to blunt the hemodynamic response to tracheal extubation due to its suppressive effects on heart rate and airway reflexes.

Various drugs like esmolol, metoprolol, fentanyl, verapamil, diltiazem, nicardipine have been used from time to time to attenuate the hemodynamic responses during tracheal extubation. There are no studies so far comparing the effects of dexmedetomidine with that of lignocaine during extubation. Hence, this study is undertaken to evaluate the hemodynamic and recovery responses between dexmedetomidine and lignocaine during tracheal extubation.

AIM AND OBJECTIVES

AIM OF THE STUDY:

The purpose of this study is to analyze and compare the properties of dexmedetomidine with that of lignocaine on the hemodynamic changes and variations in the recovery profile that occur during endotracheal extubation.

OBJECTIVES OF THE STUDY:

1. To study the hemodynamic effects of dexmedetomidine and lignocaine on the patient during extubation.
2. To compare the quality of extubation of dexmedetomidine with that of lignocaine with respect to the patient's responses.
3. To study the emergence - agitation response of the patient with dexmedetomidine and lignocaine during and following the endotracheal extubation.

MATERIALS & METHODOLOGY

MATERIALS AND METHODS

SOURCE OF THE STUDY:

Patients posted for laparoscopic abdominal surgeries at Coimbatore Medical College Hospital.

DESIGN OF THE STUDY:

Prospective study.

PERIOD OF THE STUDY :

August 2014 – July 2015

SAMPLE SIZE:

100 patients.

INCLUSION CRITERIA:

1. Patients scheduled for elective laparoscopic abdominal surgeries.
2. Patients belonging to ASA PS grade I and II.
3. Patients in the age group of 20 – 45 years.
4. Duration of surgery less than 90 minutes.

EXCLUSION CRITERIA:

1. Patients with cardiovascular diseases
2. Respiratory diseases
3. Renal diseases
4. Liver diseases
5. Uncontrolled diabetes
6. Uncontrolled hypertension
7. Difficult airway – Cormack & Lehane grade 3 and 4
8. Obesity – BMI > 30
9. History of sleep apnea

METHOD OF RANDOMIZATION:

Sealed envelope method.

MATERIALS :

1. DEXTOMID 50^{TM} (manufactured by Neon Laborotories Limited) – 0.5 ml ampoule.

   Each 0.5 ml contains Dexmedetomidine hydrochloride injection that is equivalent to 50 mcg.

2. LIGNOCARD 2% ™ (manufactured bt SPM drugs private limited) - 50 ml single use vial

   Each ml contains Lignocaine hydrochloride that is equivalent to 21.3 mg, sodium chloride 6.0 mg

METHODOLOGY:

- Institutional ethical committee approval was obtained.
- Pre anaesthetic assessment of the patient was done with a complete history, physical examination and routine investigations.
- Informed written consent was obtained from all the patients.
- Age, weight, height and body mass index of the patients were noted.
- All the patients were premedicated with Inj. Glycopyrrolate 0.2 mg iv, Inj. Midazolam 1 mg iv, Inj. Ranitidine 50 mg iv and Inj. metoclopromide 4 mg iv
- Monitoring in the operation theatre included pulseoximetry, non invasive blood pressure, five lead electrocardiogram, capnography.
- Preoxygenation was done with 100 % oxygen for three minutes.
- The patient was induced with Inj. Fentanyl 2mcg / kg, Inj. Propofol 2 mg / kg, Inj. vecuronium 0.1 mg / kg and endotracheal intubation was done.

- Anaesthesia was maintained with nitrous oxide and oxygen in the ratio of 66% : 33% along with sevoflurane 0.2 – 1 % . Inj. vecuronium was repeated in the dose of 0.03 mg / kg for the maintainence of muscle relaxation and End Tidal Carbon dioxide was maintained between 35 – 40 mm Hg.

- Normal saline and ringer lactate were used for volume replacement and maintainence.

- The patients were categorised into two different groups using the sealed envelope method.

GROUP	DRUG
Group D	Dexmedetomidine
Group L	Lignocaine

- Sevoflurane was cut off in both the groups before drug administration.

- The patients in Group D received dexmedetomidine infusion of 0.75 μg / kg in 100 ml of 0.9 % sodium chloride over a period of fifteen minutes before the anticipated time of extubation and the patients in group L patients received preservative free lignocaine 2 % at the dose of 1.5 mg / kg bolus intravenously, two minutes before the time of extubation.

- Reversal from the neuromuscular blocakade was done with Inj. Glycopyrrolate 10 mcg / kg and Inj. Neostigmine 50 mcg / kg and the trachea was extubated when the spontaneous breathing efforts were adequate and the patient obeyed commands.

- Initial parameters like the heart rate, systolic arterial blood pressure, diastolic arterial blood pressure and mean arterial pressure were documented in both the groups during injection. Later at one, three, five, ten and fifteen minutes after injection in group D and one minute after injection in group L prior to extubation.

- The same were documented during extubation and at one, three, five minutes following endotracheal extubation and after that every five minutes for upto thirty minutes in both the groups D and L.

- Extubation response was analyzed on a five point score (Extubation quality score) based on the patient's comfort and response.

EXTUBATION QUALITY SCORE	EXTUBATION RESPONSE
1	Patient is having no cough
2	Endotracheal extubation is smooth and the patient is having cough - one or two times (minimal)
3	Patient is having cough - three or four times (moderate)
4	Patient is having cough - five to ten times (severe)
5	Patient is having cough - more than ten times or laryngospasm or breath holding. Extubation is poor and the patient is restless.

- Emergence - agitation was analyzed on a six point score based on the patient's response.

EMERGENCE–AGITATION SCORE	RESPONSE OF THE PATIENT
1	The patient is apprehensive, restless or agitated.
2	The patient is calm, oriented and co – operative.
3	The patient is drowsy but is responding to commands.
4	The patient is somnolent but responds quickly to auditory or tactile stimuli.
5	The patient is somnolent, responds slowly to auditory or tactile stimuli.
6	The patient is somnolent and does not respond to auditory or tactile stimuli.

Any adverse event like vomiting, respiratory depression, laryngospasm, bronchospasm was recorded.

All data analysis were performed by using computer software SPSS of windows and P value less than 0.05 was considered as statistically significant.

OBSERVATION & RESULTS

OBSERVATIONS AND RESULTS

This study was performed on ASA PS I and II patients belonging to the age group of 20 – 45 years who were posted for laparoscopic abdominal surgeries. A comparative study of dexmedetomidine with intravenous lignocaine on hemodynamic variations and recovery profiles were carried out on 100 patients who were categorised into 2 groups of 50 each.

All data were complied, tabularized and formulated as mean +/- standard deviation. All data that were collected were compared using independent 't' test. A statistically significant difference was concluded if the value of $P < 0.05$.

The compiled results are depicted below :

TABLE 1: DEMOGRAPHIC DETAILS OF THE STUDY

SEX	Dexmedetomidine	Lignocaine
MALE	27(54%)	21(42%)
FEMALE	26(42%)	29(58%)

Table 1 shows that in the Dexmedetomidine group 54 % of them were males and 42 % were females whereas in the lignocaine group, 42 % of them were males and 58% were females.

CHART 1

DEMOGRAPHIC DETAILS

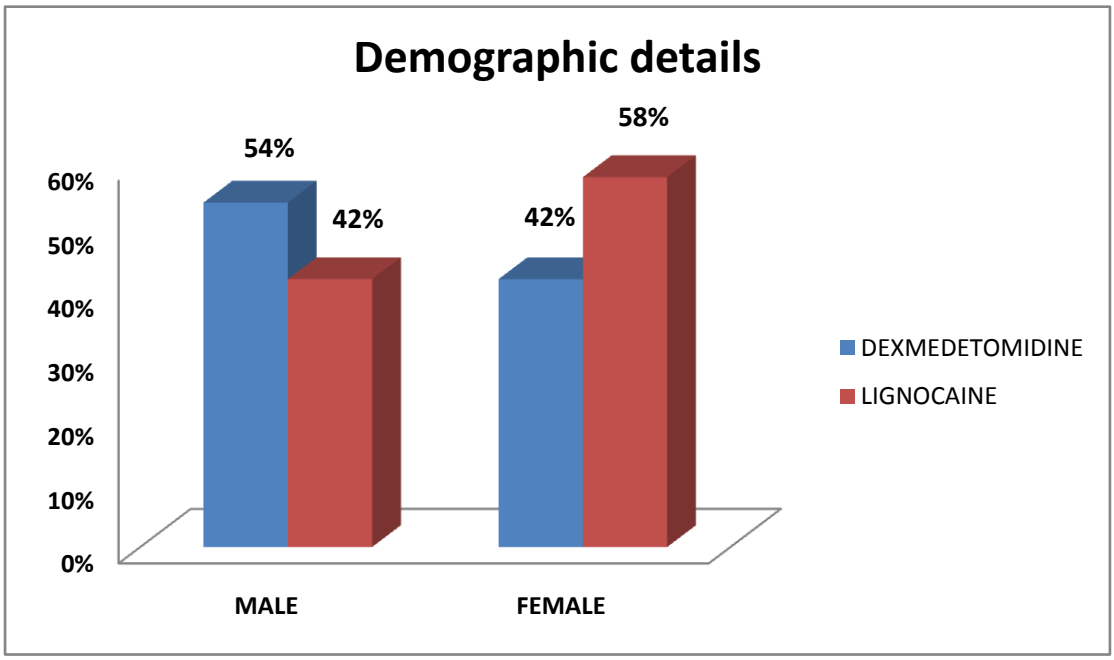


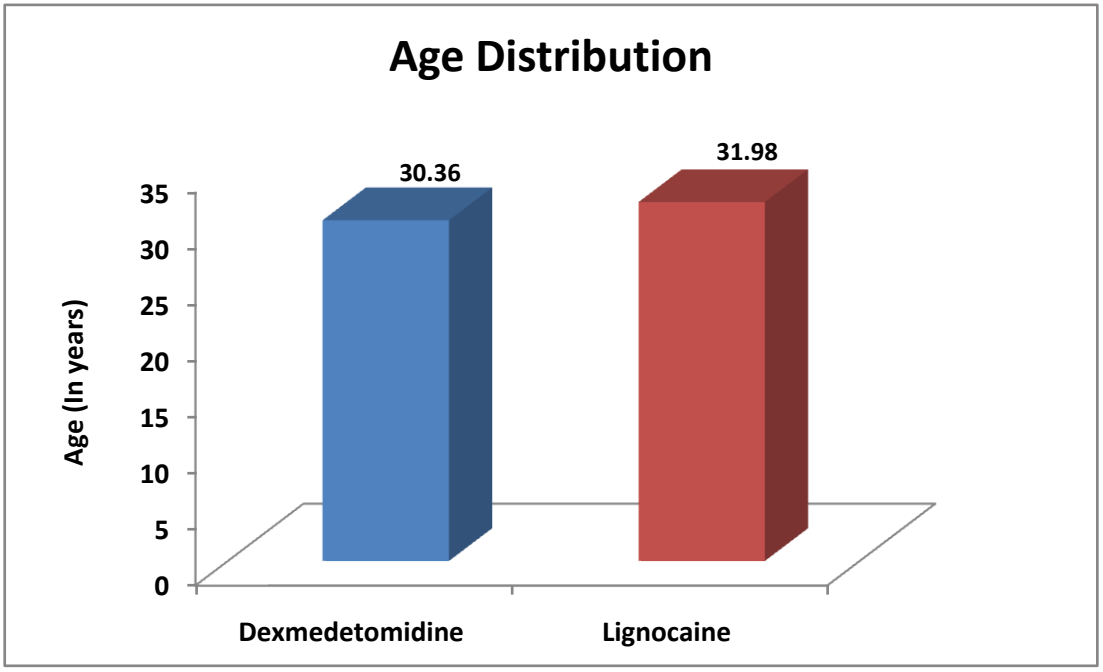
TABLE 2: DEMOGRAPHIC PROFILE : AGE DISTRIBUTION

Group	N	Mean ± SD	P Value
Dexmedetomidine	50	30.36 ± 7.8	0.978 **Not significant**
Lignocaine	50	31.98 ± 7.9	

Table 2 shows that the mean age in group Dexmedetomidine is 30.36 ± 7.8 and the mean age in group lignocaine is 31.98 ± 7.9. The P value is > 0.05 and hence, it is not statistically significant.

CHART 2

AGE DISTRIBUTION



From tables 1 and 2:

The demographic variables between the two groups Dexmedetomidine and Lignocaine were comparable. The difference in the mean values of both the groups were only minor and is not statistically significant.

TABLE 3: HEART RATE

Time in Minutes	Dexmedetomidine MEAN ± SD	Lignocaine MEAN ± SD	P Value
0	82.60 ± 13.1	81.56 ± 8.4	0.482
1	81.96 ± 12.0	88.13 ± 8.3	0.146
3	78.00 ± 11.6		
5	75.64 ± 8.9		
10	74.60 ± 9.4		
15	78.16 ± 10.6		
Extubation	91.58 ± 9.8	105.04 ± 7.7	0.041
1	93.62 ± 9.2	103.64 ± 8.1	0.005
3	88.54 ± 7.8	102.66 ± 9.8	0.026
5	84.20 ± 7.1	99.72 ± 9.5	0.030
10	79.80 ± 6.4	94.06 ± 8.7	0.039
15	77.62 ± 6.1	88.68 ± 8.1	0.046
20	72.48 ± 5.3	83.56 ± 8.0	0.003
25	69.26 ± 4.4	78.72 ± 8.0	0.000
30	65.82 ± 4.5	73.90 ± 8.1	0.000

The mean heart rate before extubation, during extubation and after extubation was calculated in both the groups, dexmedtomidine and lignocaine.

The mean heart rate before extubation in the dexmedetomidine group was 82.60 ± 13.1 and 81.96 ± 12.0 in the lignocaine group it was 81.56 ± 8.4 and 88.13 ± 8.3 respectively.

Statistical analysis shows a P value of heart rate before extubation as 0.482 and 0.146 which are statistically insignificant.

The mean heart rate during extubation in the dexmedetomidine group is 91.58 ± 9.8 and in the lignocaine group, it is 105.04 ± 7.7.

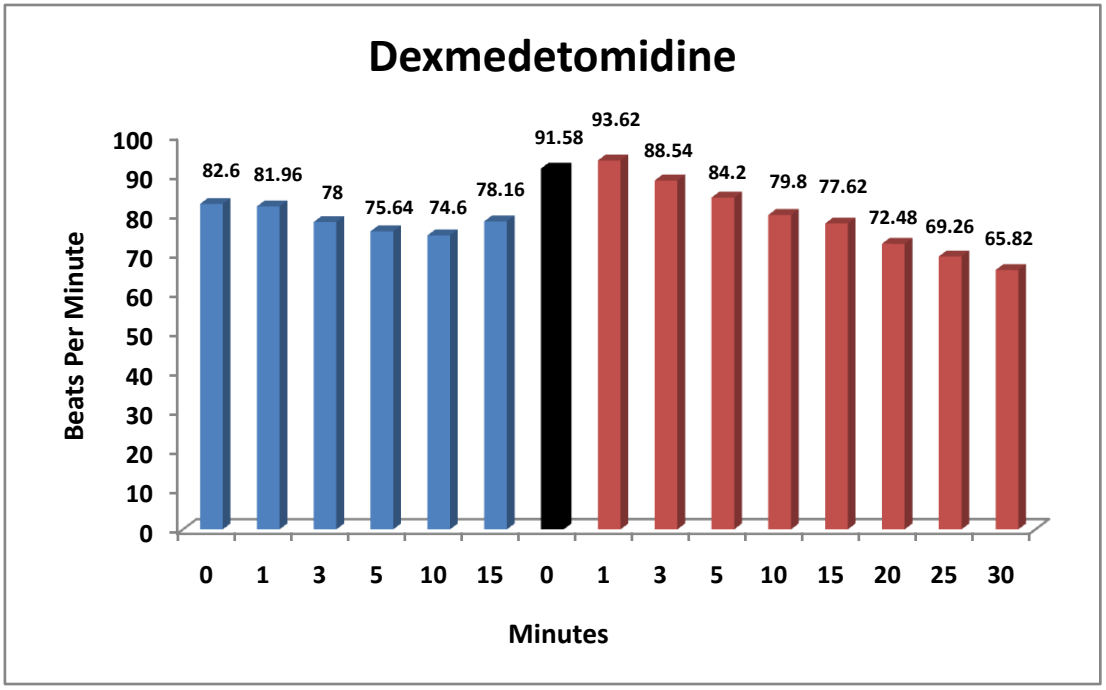
The P value of heart rate at the time of extubation is 0.041 which is statistically significant.

The mean heart rate of the dexmedetomidine group after extubation at 1, 3, 5, 10, 15, 20, 25 and 30 minutes are 93.62 ± 9.2, 88.54 ± 7.8, 84.20 ± 7.1, 79.80 ± 6.4, 77.62 ± 6.1, 72.48 ± 5.3 , 69.26 ± 4.4, 65.82 ± 4.5 respectively. The mean heart rate of the lignocaine group after extubation at 1, 3, 5, 10, 15, 20, 25 and 30 minutes are 103.64 ± 8.1, 102.66 ± 9.8, 99.72 ± 9.5, 94.06 ± 8.7 , 88.68 ± 8.1, 83.56 ± 8.0, 78.72 ± 8.0, 73.90 ± 8.1 respectively.

Statistical analysis reveals a P value of heart rate after extubation at 1, 3, 5, 10, 15, 20, 25, 30 minutes as 0.005, 0.026, 0.030, 0.039, 0.046, 0.003, 0.000, 0.000. These P values are statistically significant.

CHART 3

HEART RATE



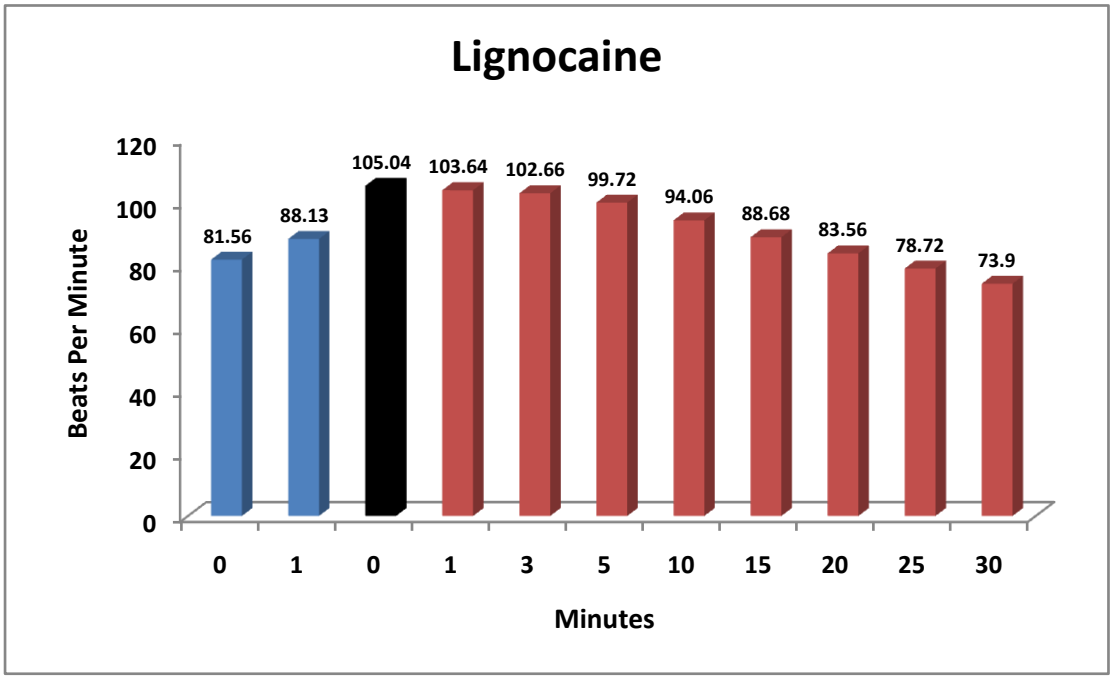


TABLE 4: SYSTOLIC BLOOD PRESSURE

Time in Minutes	**Dexmedetomidine MEAN ± SD Systolic BP**	**Lignocaine MEAN ± SD Systolic BP**	**P Value**
0	126.92 ± 10.3	121.52 ±5.5	0.270
1	126.30 ± 6.3	127.36 ±5.4	0.237
3	121.98 ± 6.1		
5	118.64 ± 6.9		
10	116.72 ± 7.0		
15	121.02 ± 7.7		
Extubation	131.3 ± 6.3	138.48 ± 4.8	0.006
1	127.96 ± 6.9	139.78 ± 4.5	0.000
3	124.10 ± 6.1	135.62 ± 4.4	0. 020
5	119.96 ± 5.5	130.78 ± 4.4	0.050
10	117.41 ± 4.8	125.16 ± 4.4	0.012
15	114.14 ± 6.1	121.00 ± 4.5	0.030
20	107.56 ± 5.3	116.18 ± 4.9	0.029
25	103.16 ± 5.7	111.06 ± 5.5	0.019
30	98.66 ± 6.1	105.74 ± 4.9	0.043

The mean systolic blood pressure of the dexmedetomidine group before extubation were126.92 ± 10.3 and 126.30 ± 6.3 whereas in the lignocaine group, it was 121.52 ± 5.5 and 127.36 ± 5.4.

The P values of the systolic blood pressure before extubation were 0.270 and 0.237 which are not statistically significant.

The mean systolic blood pressure of the dexmedetomidine group at the time of extubation was 131.3 ± 6.3 and that of lignocaine group is 138.48 ± 4.8. The P value at the time of extubation was 0.006 which is statistically significant.

The mean systolic blood pressure of the dexmedetomidine group after extubation at 1, 3, 5, 10, 15, 20, 25, 30 minutes were 127.96 ± 6.9, 124.10 ± 6.1, 119.96 ± 5.5, 117.41 ± 4.8, 114.14 ± 6.1, 107.56 ± 5.3, 103.16 ± 5.7, 98.66 ± 6.1 respectively.

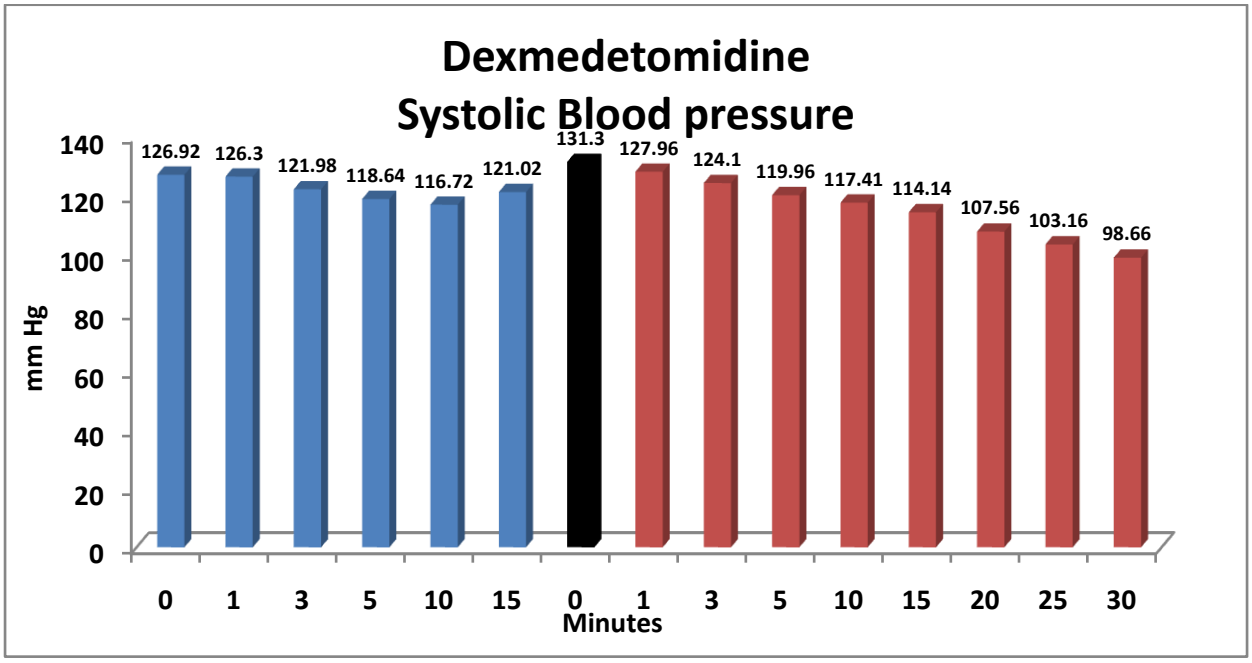
The mean systolic blood pressure of the lignocaine group after extubation at 1, 3, 5, 10, 15, 20, 25, 30 minutes were 138.48 ± 4.8, 139.78 ± 4.5, 135.62 ± 4.4, 130.78 ± 4.4, 125.16 ± 4.4, 121.00 ± 4.5, 116.18 ± 4.9, 111.06 ± 5.5, 105.74 ± 4.9

Statistical analysis reveals a P value of the systolic blood pressure after extubation at 1, 3, 5, 10, 15, 20, 25, 30 minutes were 0.000, 0.020,

0.050, 0.012, 0.030, 0.029, 0.019, 0.043 respectively. These P values are statistically significant.

CHART 4

SYSTOLIC BLOOD PRESSURE



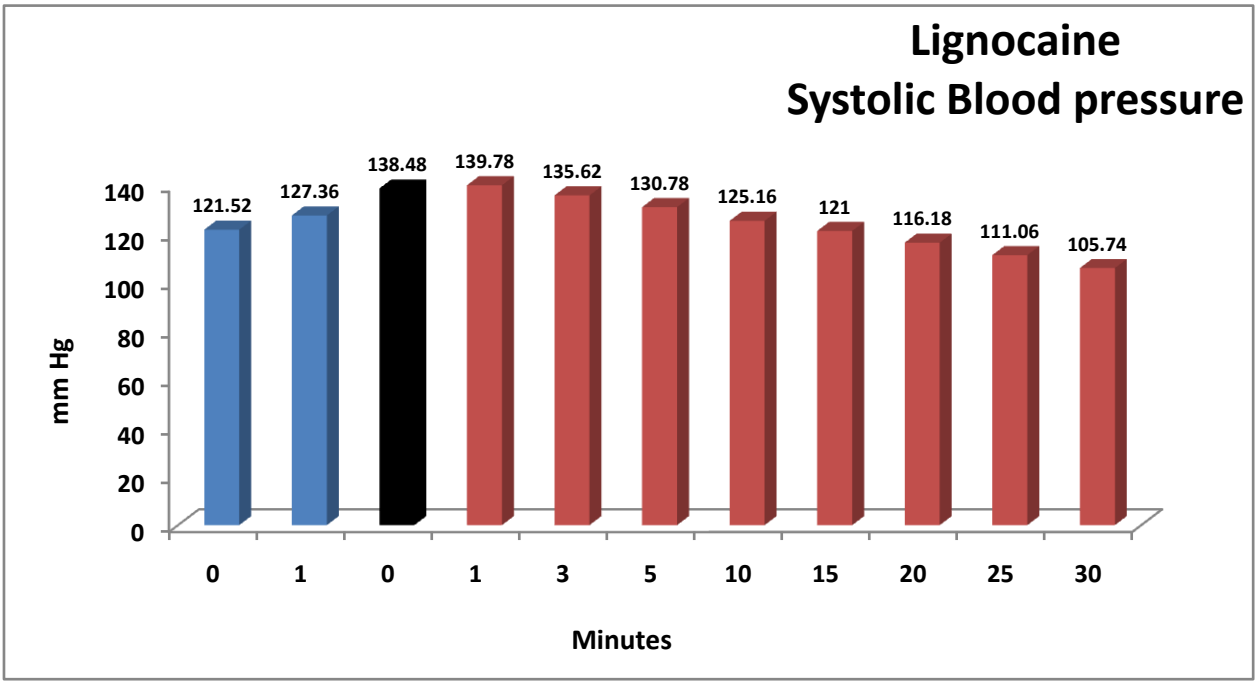


TABLE 5: DIASTOLIC BLOOD PRESSURE

Time in Minutes	Dexmedetomidine MEAN ± SD Diastolic BP	Lignocaine MEAN ± SD Diastolic BP	P Value
0	84.94 ± 5.2	77.48 ± 5.1	0.235
1	82.52 ± 3.9	83.14 ± 5.5	0.221
3	80.10 ± 4.5		
5	76.92 ± 5.1		
10	75.98 ± 4.8		
15	79.00 ± 4.4		
Extubation	87.2 ± 2.8	93.76 ± 4.0	0.042
1	84.54 ± 3.6	93.76 ± 4.0	0.032
3	81.52 ± 3.9	91.06 ± 4.6	0.049
5	77.54 ± 3.9	85.32 ± 4.3	0.036
10	75.88 ± 3.9	83.54 ± 4.3	0.050
15	73.88 ± 3.9	79.86 ± 4.7	0.094
20	68.00 ± 3.3	75.78 ± 5.1	0.001
25	64.94 ± 3.1	71.70 ± 5.2	0.000
30	61.24 ± 3.3	67.26 ± 6.0	0.001

The mean diastolic blood pressure of the dexmedetomidine group before extubation were 84.94 ± 5.2 and 82.52 ± 3.9 whereas in the lignocaine group, it was 77.48 ± 5.1 and 83.14 ± 5.5.

The P values of the diastolic blood pressure before extubation were 0.235 and 0.221 which are not statistically significant.

The mean diastolic blood pressure of the dexmedetomidine group at the time of extubation was 87.2 ± 2.8 and that of lignocaine group is 93.76 ± 40. The P value at the time of extubation was 0.042 which is statistically significant.

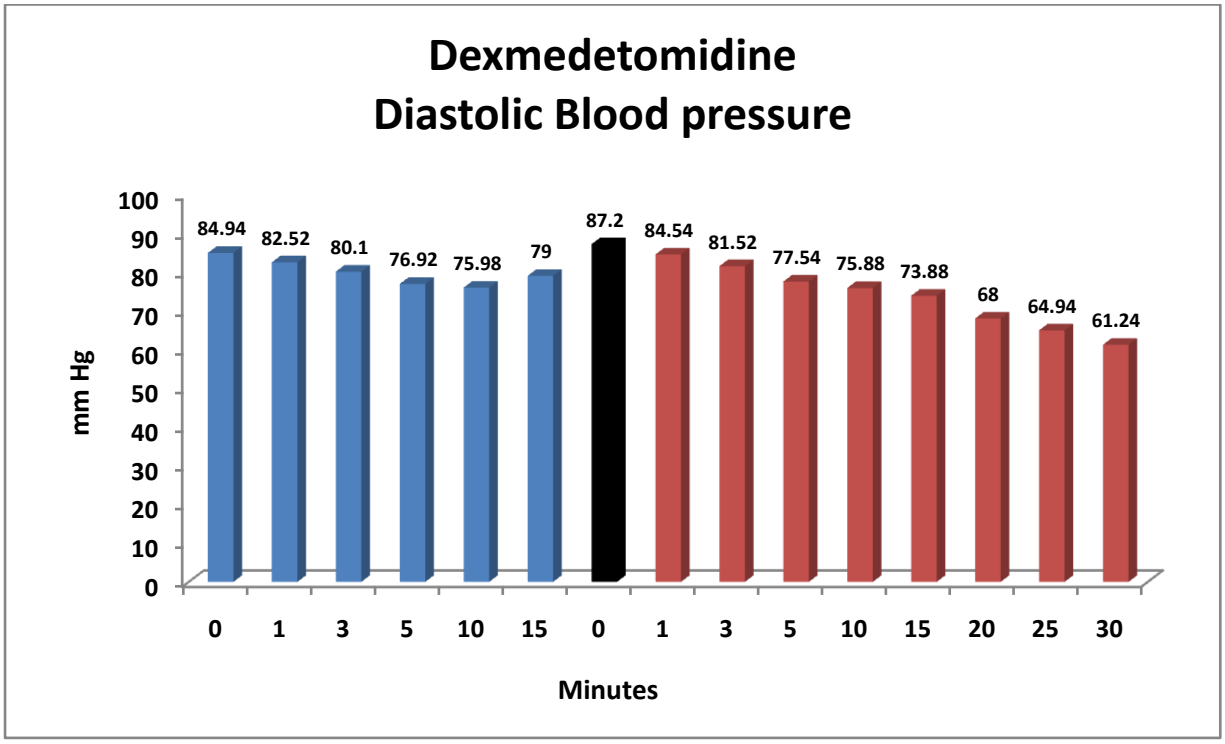
The mean diastolic blood pressure of the dexmedetomidine group after extubation at 1, 3, 5, 10, 15, 20, 25, 30 minutes were 84.54 ± 3.6, 81.52 ± 3.9, 77.54 ± 3.9, 75.88 ± 3.9, 73.88 ± 3.9, 68.00 ± 3.3, 64.94 ± 3.1, 61.24 ± 3.3 respectively.

The mean diastolic blood pressure of the lignocaine group after extubation at 1, 3, 5, 10, 15, 20, 25, 30 minutes were 93.76 ± 4.0, 93.76 ±4.0, 91.06 ± 4.6, 85.32 ± 4.3, 83.54 ± 4.3, 79.86 ± 4.7, 75.78 ± 5.1, 71.70 ± 5.2, 67.26 ± 6.0.

Statistical analysis reveals a P value of the diastolic blood pressure after extubation at 1, 3, 5, 10, 15, 20, 25, 30 minutes were 0.032, 0.049, 0.036, 0.050, 0.094, 0.001, 0.000, 0.001 respectively. These P values are statistically significant.

CHART 5

DIASTOLIC BLOOD PRESSURE



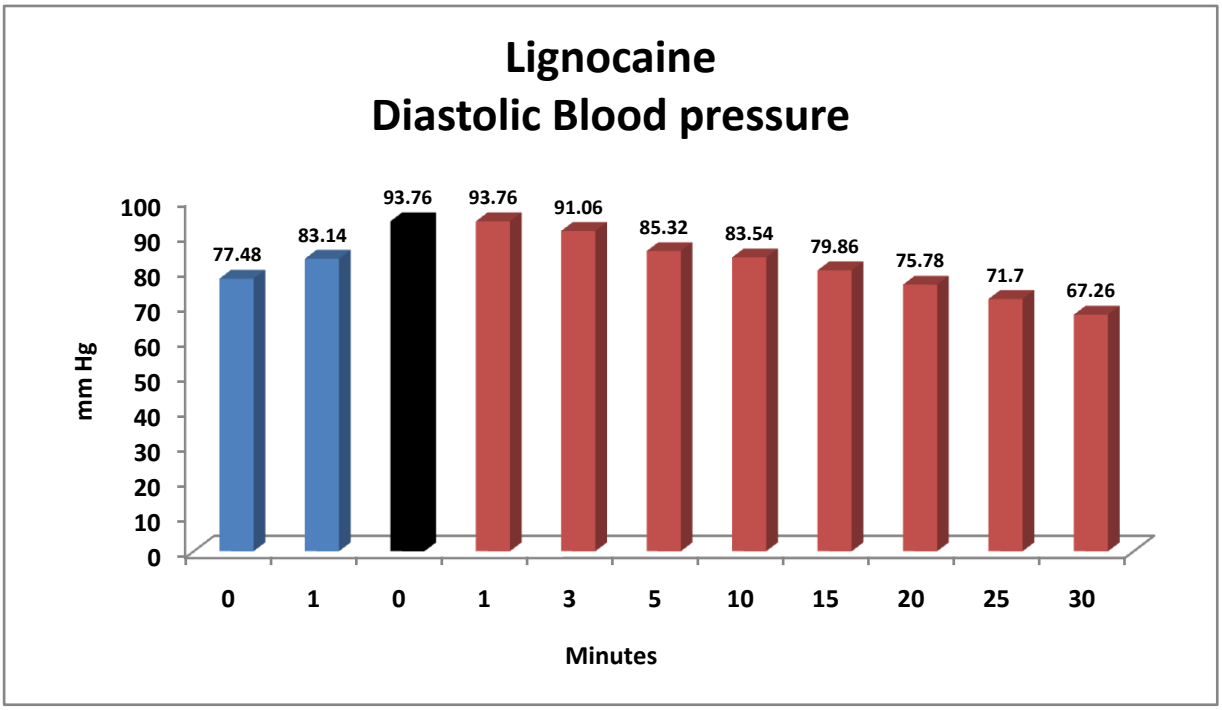


TABLE 6: MEAN ARTERIAL PRESSURE

Time in Minutes	Dexmedetomidine MEAN ± SD Mean BP	Lignocaine MEAN ± SD Mean BP	P Value
0	98.92 ± 6.1	92.14 ± 4.9	0.730
1	97.60 ± 4.3	97.84 ± 5.0	0.272
3	94.06 ± 4.6		
5	90.82 ± 4.9		
10	89.60 ± 4.6		
15	93.00 ± 4.3		
Extubation	101.84 ± 3.4	105.72 ± 3.8	0.050
1	98.94 ± 4.1	104.72 ± 3.8	0.032
3	95.74 ± 3.9	102.90 ± 4.4	0.031
5	90.68 ± 3.7	99.78 ± 4.1	0.033
10	88.72 ± 3.5	97.76 ± 4.0	0.012
15	85.29 ± 3.5	93.58 ± 4.1	0.038
20	81.18 ± 3.4	89.22 ± 4.5	0.015
25	77.64 ± 3.4	84.78 ± 4.9	0.042
30	73.62 ± 3.6	80.10 ± 5.6	0.007

The mean values of the mean arterial pressure of the dexmedetomidine group before extubation were 98.92 ± 6.1 and 97.60 ± 4.3 whereas in the lignocaine group, it was 92.14 ± 4.9 and 97.84 ± 5.0.

The P values of the mean arterial pressure before extubation were 0.730 and 0.272 which are not statistically significant.

The mean value of the mean arterial pressure of the dexmedetomidine group at the time of extubation was 101.84 ± 3.4 and that of lignocaine group is 105.72 ± 3.8 . The P value at the time of extubation was 0.050 which is statistically significant.

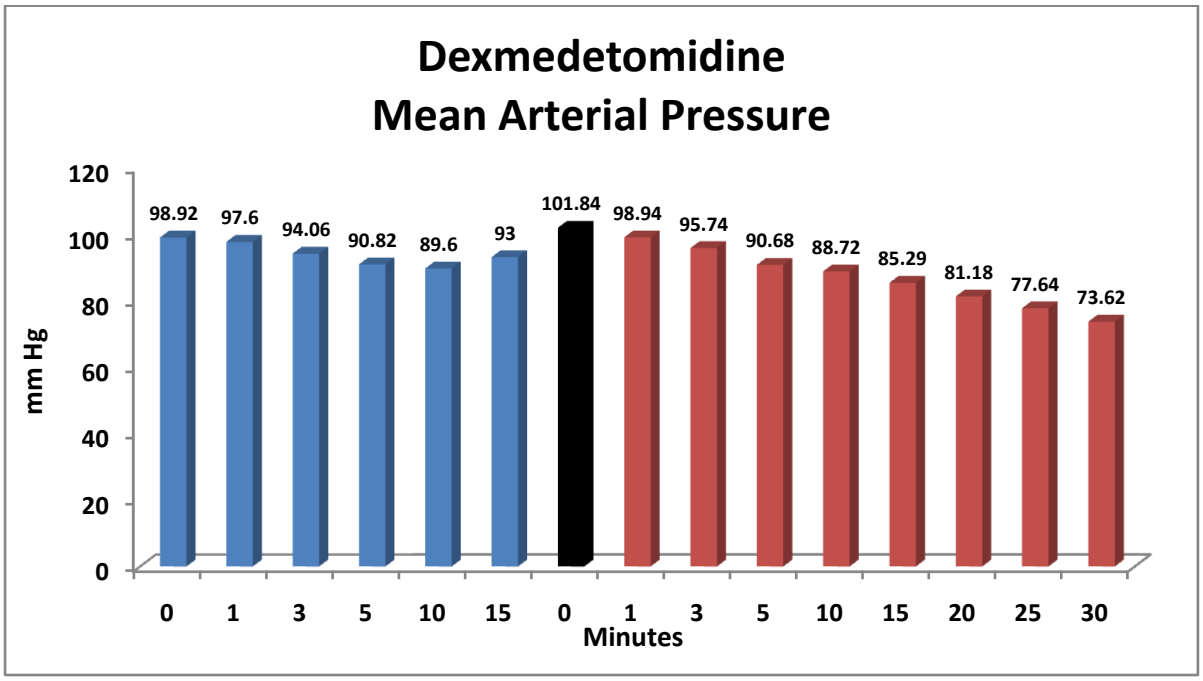
The mean values of the mean arterial pressure of the dexmedetomidine group after extubation at 1, 3, 5, 10, 15, 20, 25, 30 minutes were 98.94 ± 4.1, 95.74 ± 3.9, 90.68 ± 3.7, 88.72 ± 3.5, 85.29 ± 3.5, 81.18 ± 3.4, 77.64 ± 3.4, 73.62 ± 3.6 respectively.

The mean values of the mean arterial pressure of the lignocaine group after extubation at 1, 3, 5, 10, 15, 20, 25, 30 minutes were 104.72 ± 3.8, 102.90 ± 4.4, 99.78 ± 4.1, 97.76 ± 4.0, 93.58 ± 4.1, 89.22 ± 4.5, 84.78 ± 4.9, 80.10 ± 5.6 respectively.

Statistical analysis reveals a P value of the mean arterial pressure after extubation at 1, 3, 5, 10, 15, 20, 25, 30 minutes were 0.032, 0.031, 0.033, 0.012, 0.038, 0.015, 0.042, 0.007 respectively. These P values are statistically significant.

CHART 6

MEAN ARTERIAL PRESSURE



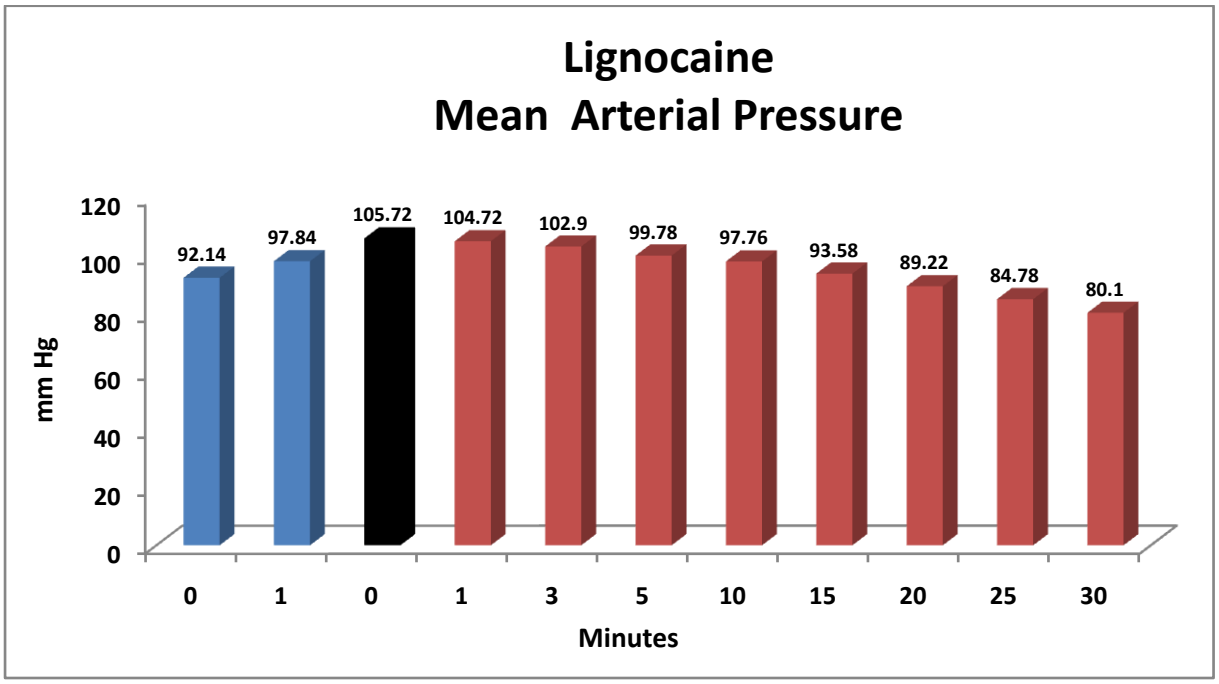


TABLE 7: EXTUBATION QUALITY SCORE

Quality score	Dexmedetomidine	Lignocaine	Total	P Value
1	6	0	6	0.048
2	36	9	45	
3	8	38	46	
4	0	3	3	
5	0	0	0	
Total	50	50	100	

From the Table 7, in the Dexmedetomidine group, 12 % of the patients had no cough during extubation whereas 72 % of the patients were extubated smoothly with minimal cough and 16 % of them had moderate cough.

In the lignocaine group, 18% of the patients had minimal cough during extubation and 76 % patients had moderate cough. 6% of the patients had severe cough.

Statistical analysis of the Extubation Quality score shows a P value of 0.048 which is statistically significant.

CHART 7

EXTUBATION QUALITY SCORE

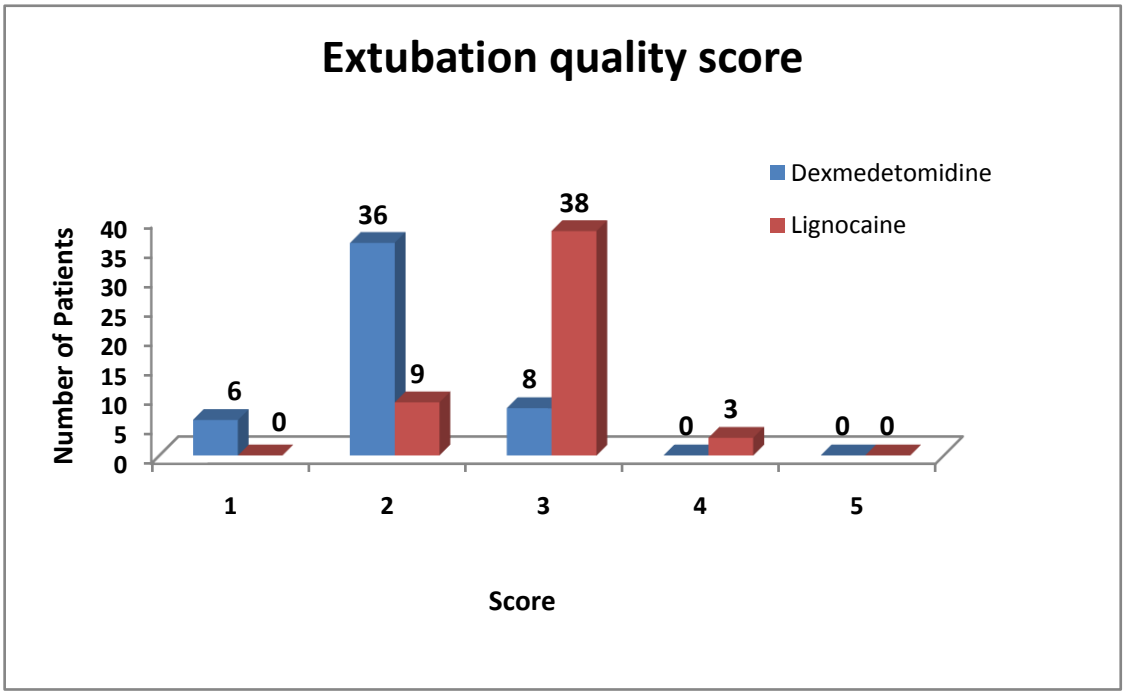


TABLE 8: EMERGENCE - AGITATION SCORE

Agitation score	Dexmedetomidine	Lignocaine	Total	P value
1	0	3	2	
2	18	42	69	
3	32	5	37	
4	0	0	2	0.032
5	0	0	0	
6	0	0	0	
Total	50	50	100	

From table 8, in the dexmedetomidine group, 36 % of the patients were calm, oriented and co- operative in the dexmedetomidine group and 64 % of the patients were drowsy, but responded to commands.

In the lignocaine group, 84 % of the patients were calm, oriented and co – operative and 10 % of the patients were drowsy and responded to commands and 6 % of the patients were apprehensive and restless.

The P value of the Emergence – Agitation score is 0.032 which is less than 0.05 and hence, it is statistically significant.

CHART 8

AGITATION SCORE

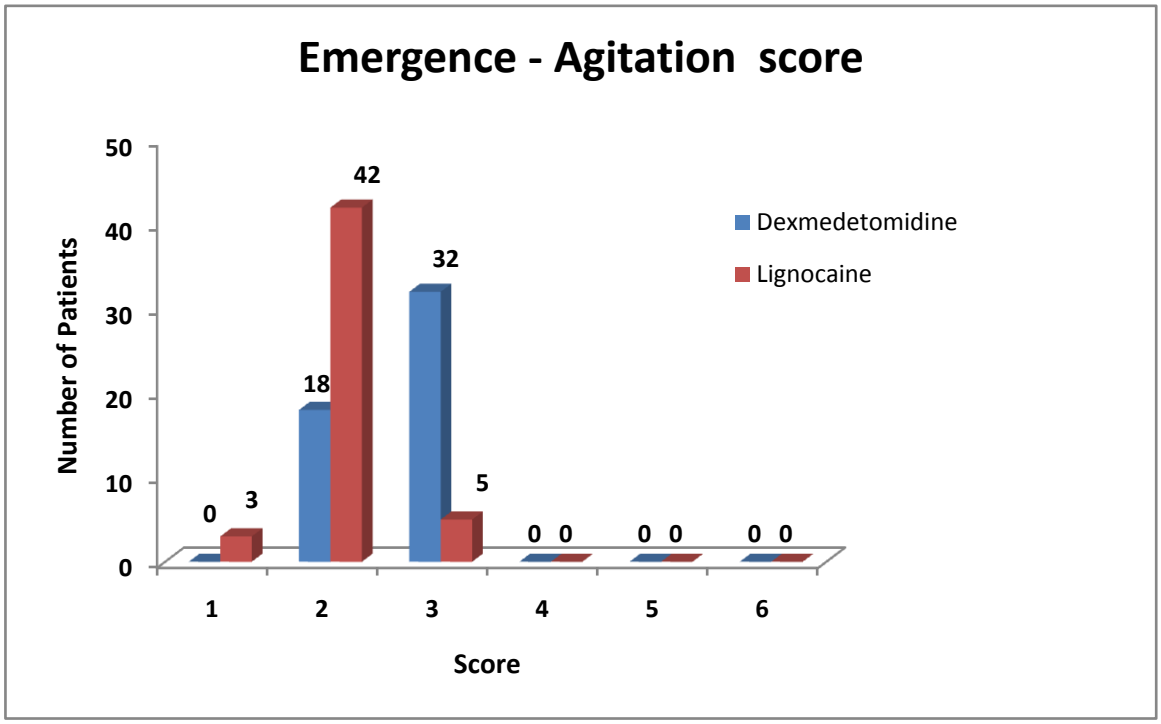


TABLE : 9 ADVERSE EFFECTS

ADVERSE EFFECTS	Dexmedetomidine	Lignocaine
Vomiting	1	3
Respiratory Depression	0	0
Laryngospasm	0	0
Bronchospasm	0	0
Bradycardia (HR<60/min)	6	1
Hypotension (MAP < 60 mm Hg)	0	0

One patient in the dexmedetomidine group had vomiting whereas 3 patients in the lignocaine group had vomiting.

None of the patient had respiratory depression, laryngospasm or bronchospasm.

6 patients had bradycardia (heart rate < 60 / min) in the dexmedetomidine group whereas, one patient had bradycardia in the lignocaine group but none of them required intervention.

The incidence of hypotension (MAP < 60 mm Hg) was not observed in any of the patients in both the groups.

DISCUSSION

DISCUSSION

Extubation of the trachea is associated with wide fluctuations in the hemodynamics that can lead to tachycardia, hypertension and arrythmias. It is also associated with reflex increases in airway reactivity leading to stress responses and airway irritation. Dexmedetomidine and lignocaine are associated with control of these hemodynamic changes and stressful airway responses. This study was undertaken to analyze the effects of dexmedetomidine versus lignocaine variations in hemodynamics and recovery responses during tracheal extubation. This study was conducted in100 patients belonging to ASA I AND II, between the age group of 20 to 45 years of age who were posted for laparoscopic abdominal surgeries.

HEART RATE

Barkha Bindu et al., compared the effects of dexmedetomidine with placebo in fifty patients of twenty five in each group. They found a statistically significant difference in the heart rate beginning from ten minutes from the time of dexmedetomidine administration which continued till twenty minutes after extubation.

In my study, the heart rate got increased in both the groups, dexmedetomidine and lignocaine as compared to their baseline values. But, the use of dexmedetomidine was associated with a lesser increase in heart

rate as compared to that of lignocaine. The heart rate variations between dexmedetomidine and lignocaine were statistically significant from the time of extubation and it continued after extubation till the time , the observations were recorded.

SYSTOLIC BLOOD PRESSURE

Barkha Bindu et al., observed that the systolic blood pressure varies statistically significant between dexmedetomidine and placebo starting from ten minutes after the drug administration and it continued till the observations were made.

In my study, the systolic blood pressure variations between dexmedetomidine and lignocaine were statistically significant from the time of extubation and it continued after extubation till the time, the observations were recorded. So, dexmedetomidine controlled systolic blood pressure better than lignocaine.

The findings are similar to the above mentioned study.

DIASTOLIC BLOOD PRESSURE

Barkha Bindu et al., observed that the diastolic blood pressure showed a statistically significant difference between dexmedetomidine and placebo starting from ten minutes after the drug administration which continued till the observations were made.

In my study, the diastolic blood pressure variations between dexmedetomidine and lignocaine were statistically significant from the time of extubation and it continued after extubation till the time, the observations were recorded.

So, the findings are concurrent with the above mentioned study.

MEAN ARTERIAL PRESSURE

Barkha Bindu et al., observed that the mean arterial blood pressure varied statistically significant between dexmedetomidine and placebo starting from ten minutes after the drug administration and it continued till the observations were made.

In my study, the mean arterial pressure got elevated in both the groups dexmedetomidine and lignocaine. But, the rise was more with the lignocaine group. It was observed that dexmedetomidine had a better control over mean arterial pressure as compared to that of lignocaine and it remained statistically significant from the time of extubation till the obswervations were made.

So, the findings are similar to the above mentioned study.

EXTUBATION QUALITY SCORE

Barkha Bindu et al., observed in their study that the quality of extubation was better in the dexmedetomidine group. 84 % patients in the

group dexmedetomidine, had minimal cough, whereas 16% patients had moderate cough during extubation. In the placebo group, 84 % patients had moderate cough during extubation, whereas only 16 % patients had minimal cough.

In my study, 72 % of the patients were extubated smoothly in the dexmedetomidine group with minimal cough whereas only 18 % of the patients were extubated smoothly in the lignocaine group. Moreover, in the dexmedetomidine group, 16 % of the patients had moderate cough whereas, in the lignocaine group, 76% of the patients had moderate cough during extubation.

Hence, in my study, the quality of extubation is better with dexmedetomidine when compared with lignocaine which is in concurrence with the study of Barkha Bindu et al.

EMERGENCE – AGITATION SCORE :

Barkha Bindu et al., observed in their study that 84% of the patients in the dexmedetomidine group were drowsy, but responded to commands; whereas, in the placebo group, 80 % of the patients were oriented, cooperative and tranquil.

In my study, 36 % of the patients were calm, oriented and co-operative in the dexmedetomidine group and 64 % of the patients were

drowsy, but responded to commands, whereas in the lignocaine group, 84 % of the patients were calm, oriented and co – operative and 10 % of the patients were drowsy and responded to commands and 6 % of the patients were apprehensive and restless. This shows that the emergence - agitation during extubation and the quality of sedation in the postoperative period is better with the dexmedetomidine group.

ADVERSE EFFECTS

In the study conducted by Barkha Bindu et al., the occurrence of bradycardia was more in the dexmedetomidine group than the placebo group. 42 % of the patients in the dexmedetomidine group had bradycardia , whereas, in the control group, 8 % had bradycardia , but treatment was not required in any of the patients. 8 % of the patients in the dexmedetomidine group developed hypotension; whereas, no patients in the placebo group had hypotension.

In my study, 12 % of the patients in the dexmedetomidine group and 2 % of the patients in the lignocaine group developed bradycardia respectively, but none of them required intervention. In my study, hypotension was not observed in any patients.

SUMMARY

SUMMARY

This is a prospective randomized comparative study of dexmedetomidine versus lignocaine on the hemodynamic and recovery responses during tracheal extubation. This study was carried out in 100 patients belonging to ASA I & II, aged between 20 and 45 undergoing laparoscopic abdominal surgeries. These hundred patients were allocated into two groups – group D for Dexmedetomidine and group L for Lignocaine with fifty in each group.

The conclusion deduced from the study are:

1. There were no significant difference between the two groups in the demographic profiles.
2. Patients in the dexmedetomidine group had a lesser increase in heart rate during and after extubation as compared to lignocaine which is statistically significant.
3. Patients in the dexmedetomidine group had a better control of systolic arterial pressure as compared to lignocaine during and after extubation which is statistically significant.
4. Patients in the dexmedetomidine group are associated with a lesser rise in diastolic blood pressure during and after tracheal extubation as compared to lignocaine which is statistically significant.

5. There is a statistically significant difference in the mean arterial pressure between dexmedetomidine and lignocaine. The dexmedetomidine group is associated with a better control in the mean arterial pressure at the time extubation and the period following extubation.
6. The Quality of Extubation was observed to be better with the dexmedetomidine group as compared with the lignocaine group and is statistically significant.
7. The Emergence – Agitation score was observed to be better with the dexmedetomidine group as compared to the lignocaine group and is statistically significant.
8. The incidence of bradycardia was observed to be more with the dexmedetomidine group as compared to the lignocaine group, but none of them required intervention.
9. The incidence of vomiting was observed to be less with the dexmedetomidine group as compared to that of the lignocaine group.
10. The other complications like hypotension, respiratory depression, bronchospasm and laryngospasm were not observed in any of the patients in both the groups.

CONCLUSION

CONCLUSION

To conclude, Dexmedetomidine administered before tracheal extubation, was more effective in maintaining the hemodynamic stability, facilitated smooth tracheal extubation and had a better quality of recovery as compared to lignocaine.

ANNEXURES

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BIBLIOGRAPHY

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PROFORMA

PROFORMA

"A COMPARATIVE STUDY ABOUT THE EFFECT OF DEXMEDETOMIDINE VERSUS LIGNOCAINE ON HEMODYNAMIC AND RECOVERY RESPONSES DURING TRACHEAL EXTUBATION"

Name :

Age :

Sex :

IP No :

ASA Status :

BMI :

Diagnosis :

Surgery :

Date of Surgery :

PRE-OP EVALUATION :

HISTORY :

GENERAL EXAMINATION :

Pallor : Icterus : Cyanosis: Clubbing:

Edema: Lymphadenopathy:

PR: BP: RR:

SYSTEMIC EXAMINATION

CVS: CNS:

RS: P/A:

INVESTIGATIONS

Blood HB% :

FBS/RBS : Urea : Creatinine :

Chest X-Ray : ECG :

GROUP D : DEXMEDETOMIDINE

TIME OF DRUG ADMINISTRATION (MINUTE)	HEART RATE (PER MINUTE)	SYSTOLIC BLOOD PRESSURE (mm Hg)	DIASTOLIC BLOOD PRESSURE (mm Hg)	MEAN ARTERIAL PRESSURE (mm Hg)
BEFORE				
0				
1				
3				
5				
10				
15				
DURING				
AFTER				
1				
3				
5				
10				
15				
20				
25				
30				

EXTUBATION QUALITY SCORE	EXTUBATION RESPONSE
1	Patient is having no cough
2	Endotracheal extubation is smooth and the patient is having cough - one or two times (minimal)
3	Patient is having cough - three or four times (moderate)
4	Patient is having cough - five to ten times (severe)
5	Patient is having cough - more than ten times or laryngospasm or breath holding. Extubation is poor and the patient is restless.

1	2	3	4	5

- Emergence - agitation was analyzed on a six point score based on the patient's response.

EMERGENCE–AGITATION SCORE	RESPONSE OF THE PATIENT
1	The patient is apprehensive, restless or agitated.
2	The patient is calm, oriented and co – operative.
3	The patient is drowsy but is responding to commands.
4	The patient is somnolent but responds quickly to auditory or tactile stimuli.
5	The patient is somnolent, responds slowly to auditory or tactile stimuli.
6	The patient is somnolent and does not respond to auditory or tactile stimuli.

1	2	3	4	5	6

Adverse effects :

Respiratory depression	
Vomiting	
Laryngospasm	
Bronchospasm	

GROUP L : LIGNOCAINE

TIME OF DRUG ADMINISTRATION (MINUTE)	HEART RATE (PER MINUTE)	SYSTOLIC BLOOD PRESSURE (mm Hg)	DIASTOLIC BLOOD PRESSURE (mm Hg)	MEAN ARTERIAL PRESSURE (mm Hg)
BEFORE				
0				
1				
DURING				
AFTER				
1				
3				
5				
10				
15				
20				
25				
30				

EXTUBATION QUALITY SCORE	EXTUBATION RESPONSE
1	Patient is having no cough
2	Endotracheal extubation is smooth and the patient is having cough - one or two times (minimal)
3	Patient is having cough - three or four times (moderate)
4	Patient is having cough - five to ten times (severe)
5	Patient is having cough - more than ten times or laryngospasm or breath holding. Extubation is poor and the patient is restless.

1	2	3	4	5

- Emergence - agitation was analyzed on a six point score based on the patient's response.

EMERGENCE–AGITATION SCORE	RESPONSE OF THE PATIENT
1	The patient is apprehensive, restless or agitated.
2	The patient is calm, oriented and co – operative.
3	The patient is drowsy but is responding to commands.
4	The patient is somnolent but responds quickly to auditory or tactile stimuli.
5	The patient is somnolent, responds slowly to auditory or tactile stimuli.
6	The patient is somnolent and does not respond to auditory or tactile stimuli.

1	2	3	4	5	6

Adverse effects :

Respiratory depression	
Vomiting	
Laryngospasm	
Bronchospasm	

ABBREVIATIONS

LIST OF ABBREVIATIONS USED

ABP	Arterial blood pressure
ASA	American society of Anaesthesiologists
BP	Blood pressure
C-AMP	Cyclic adenine monophosphate
CBF	Cerebral blood flow
CNS	Central Nervous System
CO2	Carbondioxide
DBP	Diastolic blood pressure
ETCO2	End tidal carbondioxide
FDA	Food and Drug Administration
GABA	Gamma Amino Butryic Acid
GI	Gastrointestinal
HR	Heart rate
ICT	Intra cranial tension
IVRA	Intravenous Regional Anaesthesia
ICU	Intensive care unit
IV	Intravenous
IM	Intramuscular
LC	Locus caeruleus
MAP	Mean Arterial Pressure
MAC	Minimum alveolar concentration

Nacl	Sodium chloride
PaO2	Partial pressure of arterial oxygen
PO2	Partial pressure of oxygen
PS	Physical Status
RLN	Recurrent Laryngeal Nerve
SLN	Superior Laryngeal Nerve
SBP	Systolic blood pressure
SD	Standard Deviation
TIVA	Total intravenous anesthesia
t ½	Half life
UO	Urine output
Vdss	Volume of distribution

CONSENT FORM

INFORMED CONSENT FORM

I am Dr. Lakshmi J, carrying out a study on the topic, "A comparative study of the effect of dexmedetomidine versus lignocaine on hemodynamic and recovery responses during tracheal extubation".

My research project is being carried out under the department of Anaesthesiology, Coimbatore Medical College and Government hospital.

RESEARCH BEING DONE:

"A COMPARATIVE STUDY OF THE EFFECT OF DEXMEDETOMIDINE VERSUS LIGNOCAINE ON HEMODYNAMIC AND RECOVERY RESPONSES DURING TRACHEAL EXTUBATION".

PURPOSE OF RESEARCH

4. To compare the hemodynamic effects of dexmedetomidine and lignocaine on the patient during extubation.
5. To evaluate the quality of extubation between dexmedetomidine and lignocaine with respect to the patient's responses.
6. To study the emergence - agitation response of the patient with dexmedetomidine and lignocaine after extubation.

SAMPLE SIZE:

100 Patients

STUDY PARTICIPANTS:

Adults with 20 - 45 years with ASA physical status I and II posted for laparoscopic abdominal surgeries.

LOCATION:

Coimbatore Medical College and Hospital, Coimbatore.

PROCEDURES INVOLVED:

The research includes detailed clinical examination including medical history, physical examination. After the initial examination, patients will be randomly allocated into either group : group D for dexmedetomidine and group L for lignocaine.

You, Shri./ Smt./ Kum. _____, aged ____ years, S/o / D/o / W/o _____, residing at _____ are requested to be a participant in the research study titled ' ***" A COMPARATIVE STUDY OF THE EFFECT OF DEXMEDETOMIDINE VERSUS LIGNOCAINE ON HEMODYNAMIC AND RECOVERY RESPONSES DURING TRACHEAL EXTUBATION"*** in Government Medical College Hospital,

Coimbatore. You satisfy eligibility criteria as per the inclusion criteria. You can ask any question or seek any clarifications on the study that you may have before agreeing to participate.

DECLINE FROM PARTICIPATION

You are hereby made aware that participation in this study is purely voluntary and honorary and that you have the option and the right to decline from participation in the study.

PRIVACY AND CONFIDENTIALITY

You are hereby assured about your privacy. Privacy of subject will be respected and any information about you or provided by you during the study will be kept strictly confidential.

AUTHORIZATION TO PUBLISH RESULTS

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified; neither will your privacy be breached.

STATEMENT OF CONSENT

I, _____, do hereby volunteer and consent to participate in this study being conducted by Dr. Lakshmi J. I have read and

understood the consent form / or it has been read and explained to me. The study has been fully explained to me, and I may ask questions at any time.

Signature / Left Thumb Impression of the Volunteer Date:

Signature and Name of witness Date:

ஒப்புதல் படிவம்

பெயர் :

வயது :

பாலினம் :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் மயக்க மருந்தவியல் மருத்துவ துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவி மரு.ஜெ.லட்சுமி அவர்கள் தனது மேற்படிப்பிற்காக மேற்கொள்ளும் ஆய்வறிக்கையாகிய " அறுவை சிகிச்சை முடிந்து செயற்கை சுவாச குழாயை நீக்கும் போது உண்டாகும் விளைவுகளை கட்டுப்படுத்த டெக்ஸ்மெடிடோமிடின் மற்றும் லிக்னொகெய்ன் மருந்துகளை ஒப்பீடு செய்தல்" பற்றி முழுவதுமாக தெரிந்து கொண்டேன். மேலும் அது சம்பந்தமான செய்முறைகள், அதனால் ஏற்படும் விளைவுகள், மற்றும் அதற்கான மருத்துவ சிகிச்சையினையும் அறிந்து கொண்டு எனது சந்தேகங்களை தெளிவுபடுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னைப் பற்றிய அனைத்து விபரங்கள் பாதுகாக்கப் படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபணை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்வில் இருந்து நான் விலகி கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

நாள் : இப்படிக்கு,

இடம் :

கையொப்பம் / ரேகை

MASTER CHARTS

MASTER CHART

DEXMEDETOMIDINE																																								
S NO	NAME	AGE	SEX	IP NO	BMI	TYPE OF SURGERY	HEART RATE (In Minutes)																BLOOD PRESSURE (In mm Hg)																		
							BEFORE EXTUBATION						DURING EXTUBATION	AFTER EXTUBATION								BEFORE EXTUBATION																				
							0	1	3	5	10	15		1	3	5	10	15	20	25	30	0 Minute			1 Minute			3 Minute			5 Minute			10 Minute			15 Minute		
																						S	D	M	S	D	M	S	D	M	S	D	M	S	D	M	S	D	M
1	Palanisamy	35	M	39545	23.2	Lap appendicectomy	90	86	80	78	86	92	102	94	90	84	76	72	70	68	66	130	86	101	128	84	99	124	80	95	120	76	91	116	72	87	126	82	97
2	Sahana	25	F	41357	20.2	Lap appendicectomy	86	84	80	76	80	82	96	102	94	88	84	82	76	74	72	118	87	97	120	84	96	116	82	93	114	76	89	108	76	87	116	82	93
3	Valliammal	45	F	39841	22.7	Lap cholecystectomy	94	96	88	84	80	86	102	98	94	90	86	80	76	72	70	128	88	101	124	84	97	120	80	93	116	76	89	112	72	85	118	84	95
4	Lakshmi	35	F	39133	24.8	Lap cholecystectomy	88	90	84	80	78	74	98	94	90	88	82	78	74	70	68	130	90	103	126	88	101	122	82	95	118	78	91	112	76	88	116	80	92
5	Kaliyappan	36	M	43517	21.2	Lap appendicectomy	96	100	94	86	78	74	92	100	94	86	82	78	76	72	68	120	86	97	122	84	97	120	80	93	116	78	91	112	76	88	120	76	91
6	Janat nisha	42	F	3064	24.1	Lap appendicectomy	94	90	84	80	76	82	96	104	94	90	86	84	80	76	70	130	88	102	126	86	99	124	82	96	120	78	92	118	74	89	112	70	84
7	Manjula	20	F	24124	22.2	lap appendicectomy	102	98	92	86	84	80	98	102	96	92	85	80	76	72	68	130	92	105	130	90	103	126	86	99	120	82	95	116	78	91	112	74	87
8	Priya	28	F	42971	23.1	Lap appendicectomy	98	96	90	86	82	78	74	92	88	84	80	75	72	68	64	128	86	100	126	82	97	122	78	93	116	74	88	112	70	84	118	76	90
9	Ravikumar	24	M	45094	24.4	Lap appendicectomy	80	78	80	78	72	72	96	94	88	84	78	75	72	68	66	136	94	108	132	92	105	120	84	96	121	82	95	114	82	93	110	76	87
10	Ramamoorthy	23	M	46480	20.8	Lap appendicectomy	86	80	76	80	84	90	102	97	91	88	84	80	78	74	70	124	82	96	126	80	95	120	76	91	116	72	87	120	76	91	122	76	91
11	Palanisamy	29	M	45979	19.2	Lap appendicectomy	88	84	80	78	76	82	96	90	86	80	77	75	72	70	68	124	86	99	120	84	96	120	80	93	118	76	90	124	82	96	132	84	100
12	Kalaiselvan	35	M	47373	23.8	Lap appendicectomy	72	74	69	67	62	65	82	86	84	80	75	72	70	66	64	138	88	105	133	86	102	126	87	100	125	82	96	120	78	92	126	74	91
13	Bhuvaneshwari	28	F	47033	22.2	Lap appendicectomy	90	88	82	80	76	72	98	102	106	98	92	88	80	74	70	136	90	105	134	84	101	130	82	98	126	80	95	120	76	91	126	80	95
14	Pushpa	45	F	44840	23.1	Lap cholecystectomy	68	64	66	62	58	64	80	76	72	74	70	66	64	60	58	126	82	97	124	84	97	120	80	93	120	76	91	114	72	86	124	80	95
15	Vanipriya	24	F	49541	19.4	Lap cholecystectomy	78	80	70	72	80	88	90	96	86	80	76	70	66	64	58	126	88	101	122	84	97	118	80	93	116	82	93	120	84	96	118	80	93
16	Akkim	32	M	57102	24.1	Lap appendicectomy	82	80	78	70	72	80	96	92	86	78	76	70	72	68	64	134	86	102	132	82	99	130	82	98	126	76	93	128	70	89	132	76	94
17	Sulochana	30	F	18631	22.3	lap appendicectomy	86	82	76	70	68	67	88	86	85	82	78	75	74	72	70	118	87	97	120	84	96	124	86	99	120	80	93	114	76	89	103	77	86
18	Abdul wahith	38	M	59091	21.2	Lap appendicectomy	68	64	60	62	76	80	86	84	78	74	72	68	66	64	58	126	83	97	113	73	86	105	66	79	96	63	74	104	67	79	116	78	91
19	Srinivasa rao	37	M	58097	20.6	Lap appendicectomy	30	76	64	60	58	64	86	80	76	72	70	68	64	60	56	140	82	101	136	80	98	130	76	94	128	72	90	126	68	87	130	70	90
20	Unnikrishnan	29	M	60628	22.5	Lap appendicectomy	60	56	54	62	68	72	90	88	85	82	76	75	72	66	62	132	90	104	134	84	101	130	80	97	128	74	92	128	72	90	132	72	92
21	Mallika	40	F	63658	20.4	Lap appendicectomy	98	94	86	82	80	76	92	86	82	82	76	72	68	70	65	128	83	98	130	81	97	122	75	91	125	85	98	110	70	83	117	66	83
22	Praveen Kumar	24	M	15695	24.4	Lap appendicectomy	88	90	84	81	78	86	102	100	96	92	88	82	78	74	70	68	66	67	125	87	100	124	84	97	120	80	93	116	78	91	122	82	95
23	Babu	35	M	24853	20.4	Lap cholecystectomy	96	92	98	88	82	90	104	106	95	90	86	82	78	76	72	130	88	102	128	86	100	122	84	97	116	80	92	112	78	89	128	86	100
24	Madhura	20	F	55940	20.8	lap appendicectomy	90	94	86	82	78	90	102	98	95	90	85	82	76	72	68	126	82	97	128	80	96	124	76	92	120	72	88	116	70	85	126	82	97
25	Visalatchi	24	F	27509	24.7	lap appendicectomy	80	78	76	72	74	72	94	92	86	82	78	70	72	70	68	134	94	107	132	90	104	120	84	96	120	86	97	114	82	93	110	76	87
26	Meenakshi	20	F	81761	19.4	lap appendicectomy	92	94	90	84	88	102	112	98	92	86	82	78	74	70	66	130	82	98	126	80	95	120	78	92	116	72	87	120	78	92	122	84	97
27	Manikandan	20	M	52612	22.5	lap appendicectomy	84	80	86	90	96	100	110	98	96	92	86	80	76	72	74	128	83	98	136	80	98	122	75	91	124	86	99	120	88	99	122	84	97
28	Kala	32	F	79819	23.2	Lap appendicectomy	80	82	76	78	74	78	96	93	88	80	76	73	68	70	65	140	80	100	136	82	100	132	80	97	128	76	93	132	82	99	130	78	95
29	Mukesh	29	M	71753	20.6	Lap cholecystectomy	102	96	92	88	84	86	96	98	92	88	84	80	78	72	68	130	80	97	126	80	95	120	78	92	116	74	88	118	80	93	120	84	96
30	Saraswathy	42	F	71791	22.5	Lap cholecystectomy	68	58	54	66	62	66	80	78	70	66	64	60	56	58	54	120	86	97	114	82	93	110	80	90	106	76	86	104	78	87	106	80	89
31	Shanmugavel	25	M	72766	23.2	lap appendicectomy	75	72	68	64	74	82	98	95	92	88	84	81	76	72	68	124	84	97	120	82	95	116	80	92	114	76	89	110	72	85	118	82	94
32	Rajan	42	M	32206	23.8	Lap cholecystectomy	64	62	58	60	56	64	82	80	80	76	72	68	65	62	64	120	78	92	120	76	91	116	74	88	112	70	84	114	76	89	126	84	98
33	Kamaladevi	33	F	75250	22.3	Lap cholecystectomy	88	86	80	76	74	78	102	106	96	92	86	80	74	72	70	134	94	107	130	90	103	126	88	101	118	84	95	116	78	91	120	84	96
34	Shobana	20	F	7015	22.2	lap appendicectomy	92	96	90	86	82	88	110	114	106	98	92	85	78	72	65	128	84	99	130	84	99	128	80	96	124	76	92	118	72	87	128	80	96
35	Srinivas	25	M	76912	23.1	lap appendicectomy	84	90	88	80	72	76	84	86	82	80	76	74	70	68	66	130	86	101	126	84	98	124	86	99	120	84	96	122	88	99	120	84	96
36	Manoharan	45	M	78535	19.4	Lap cholecystectomy	76	70	68	64	60	58	78	80	78	76	72	70	66	64	62	126	84	98	120	80	93	116	76	89	114	70	85	116	74	88	120	78	92
37	Karuppusamy	42	M	78814	22.9	Lap cholecystectomy	90	94	88	85	78	86	102	106	96	90	84	78	75	68	62	130	86	101	134	88	103	130	84	99	125	80	95	120	76	91	126	80	95
38	Muthu kumar	20	M	7147	23.1	lap appendicectomy	83	88	86	80	86	90	102	100	94	88	84	80	78	73	69	130	85	100	132	93	106	134	90	105	134	86	102	132	80	97	130	84	99
39	Saravanan	24	M	49835	20.2	Lap appendicectomy	90	94	87	82	88	94	110	114	98	94	90	85	78	72	66	118	76	90	110	72	85	106	70	82	100	68	79	98	68	78	108	76	87
40	Ganesh	31	M	80208	22.9	lap appendicectomy	78	74	70	68	64	72	90	86	85	82	76	74	70	68	65	120	80	93	118	80	93	116	76	89	110	72	85	114	74	87	126	80	95
41	Karthikeyan	26	M	33000	23.5	lap appendicectomy	86	84	80	75	80	88	104	102	95	90	86	80	75	70	66	124	76	92	126	80	95	122	76	91	116	74	88	120	78	92	126	82	97
42	Sivaranjani	23	F	38246	24.2	lap appendicectomy	98	96	90	86	82	78	74	92	86	84	80	75	72	68	64	128	86	100	126	82	97	122	78	93	116	74	88	112	70	84	118	76	90
43	Surya	22	M	40916	22.9	Lap cholecystectomy	70	64	66	64	58	62	82	85	82	78	75	72	68	65	63	126	82	97	124	84	97	120	80	93	120	76	91	114	72	86	124	80	95
44	Jitendar kumar	34	M	17996	23.1	lap appendicectomy	84	82	75	70	70	67	85	88	85	82	78	75	72	70	65	118	87	97	120	84	96	124	86	99	120	80	93	114	76	89	103	77	86
45	Ismail	27	M	41692	19.9	Lap appendicectomy	60	58	54	65	68	73	90	94	92	92	86	82	78	75	72	132	90	104	134	84	101	130	80	97	128	74	92	128	72	90	132	72	92
46	Shanmuga Lakshmi	30	F	41830	21.6	Lap cholecystectomy	94	92	97	88	84	90	104	106	96	90	86	78	82	76	72	130	88	102	128	86	100	122	84	97	116	80	92	112	78	89	128	86	100
47	Senthil kumar	26	M	47145	23.4	lap appendicectomy	84	78	76	72	74	72	94	92	88	82	78	72	70	70	68	134	94	107	132	90	104	120	84	96	120	86	97	114	82	93	110	76	87
48	Aishwarya	20	F	47053	20.2	Lap appendicectomy	80	82	78	80	74	78	96	93	88	80	76	68	73	70	65	140	80	100	136	82	100	132	80	97	128	76	93	132	82	99	130	78	95
49	Mariya Sagayarani	45	F	45905	22.7	Lap cholecystectomy	64	62	58	60	56	64	78	76	73	68	64	62	60	58	54	120	78	92	120	76	91	116	74	88	112	70	84	114	76	89	126	84	98
50	Saraswathi	32	F	53069	24.8	Lap cholecystectomy	76	70	68	69	60	58	78	82	80	78	75	72	70	68	65	126	84	98	120	80	93	116	76	89	114	70	85	116	74	88	120	78	92

DEXMEDETOMIDINE																															
BLOOD PRESSURE (In mm Hg)																												EXTUBATION QUALITY SCORE	EMERGENCE AGITATION SCALE	ADVERSE EFFECTS			
S NO	DURING EXTUBATION			AFTER EXTUBATION																										RESPIRATORY DEPRESSION	VOMITING	LARYNGOSPAS	BRONCHOSPAS
				1 Minute			3 Minute			5 Minute			10 Minute			15 Minute			20 Minute			25 Minute			30 Minute							
	S	D	M	S	D	M	S	D	M	S	D	M	S	D	M	S	D	M	S	D	M	S	D	M	S	D	M			NO	NO	NO	NO
1	130	88	102	124	84	97	122	82	95	120	76	91	116	74	88	112	72	85	108	68	81	106	66	79	100	60	73	1	3	NO	NO	NO	NO
2	122	88	99	112	78	89	110	74	86	110	68	82	108	70	83	104	68	80	102	68	79	96	66	76	90	62	71	2	3	NO	NO	NO	NO
3	126	88	101	124	82	96	120	78	92	116	74	88	112	70	84	108	68	81	104	64	77	98	62	74	94	60	71	1	2	NO	NO	NO	NO
4	120	84	96	116	80	92	112	78	89	110	74	86	108	70	83	106	68	81	102	66	78	98	62	74	96	60	72	3	3	NO	NO	NO	NO
5	128	82	97	130	84	99	120	80	93	116	78	91	112	74	87	108	70	83	106	68	81	100	64	76	94	60	71	2	3	NO	NO	NO	NO
6	128	84	99	122	80	94	120	76	91	116	76	89	112	72	85	108	70	83	104	68	80	100	64	76	96	62	73	1	2	NO	NO	NO	NO
7	120	82	95	118	80	93	116	76	89	112	72	85	110	68	82	108	66	80	102	64	77	100	62	75	96	58	71	2	3	NO	NO	NO	NO
8	126	86	99	120	84	96	116	80	92	112	76	88	108	74	85	106	70	82	102	68	79	98	64	75	92	60	71	2	3	NO	NO	NO	NO
9	120	86	97	120	84	96	118	80	93	114	76	89	110	72	85	106	67	80	104	64	77	100	60	73	97	58	71	3	2	NO	NO	NO	NO
10	130	82	98	126	80	95	120	76	91	116	72	87	110	70	83	104	66	79	100	62	75	96	60	72	92	58	69	2	3	NO	NO	NO	NO
11	140	90	107	132	84	100	130	80	97	124	76	92	119	73	88	116	72	87	108	68	81	104	70	81	100	66	77	2	3	NO	NO	NO	NO
12	136	88	104	130	82	98	126	78	94	122	74	90	120	70	87	116	70	85	112	68	83	106	64	78	100	60	73	2	3	NO	NO	NO	NO
13	140	90	107	138	90	106	136	86	103	130	80	97	124	82	96	116	78	91	118	72	87	112	68	83	110	64	79	2	2	NO	YES	NO	NO
14	138	88	105	136	88	104	130	82	98	126	78	94	120	72	88	118	70	86	114	68	83	110	64	79	112	60	77	2	3	NO	NO	NO	NO
15	126	86	99	122	84	97	120	82	95	116	78	91	112	72	85	104	68	80	100	64	76	94	62	73	90	62	71	2	3	NO	NO	NO	NO
16	140	88	105	136	86	103	130	84	99	126	84	98	120	78	92	120	74	89	116	80	92	108	70	83	104	66	79	2	2	NO	NO	NO	NO
17	124	86	99	122	84	97	120	80	93	116	76	89	112	72	85	106	68	81	102	66	78	96	63	74	90	56	67	2	3	NO	NO	NO	NO
18	126	86	99	120	80	93	116	76	89	114	72	86	110	70	83	106	66	79	100	66	77	96	62	73	92	58	69	3	3	NO	NO	NO	NO
19	140	90	107	136	86	103	132	84	100	126	76	93	118	76	90	112	74	87	110	70	83	108	66	80	104	64	77	2	3	NO	NO	NO	NO
20	140	84	102	136	80	98	130	80	97	128	76	93	122	72	89	118	74	89	114	72	86	110	70	83	102	66	78	2	3	NO	NO	NO	NO
21	130	82	98	130	80	97	125	77	93	122	74	90	120	70	87	114	68	83	110	64	79	107	60	76	100	56	71	2	2	NO	NO	NO	NO
22	132	86	101	130	84	99	126	82	97	120	80	93	116	76	89	112	73	86	110	68	82	98	66	77	96	64	75	2	3	NO	NO	NO	NO
23	124	84	97	120	80	93	116	78	91	112	74	87	110	72	85	104	68	80	100	65	77	98	64	75	94	60	71	3	3	NO	YES	NO	NO
24	134	88	103	132	86	101	126	85	99	120	80	93	115	75	88	112	74	87	106	70	82	100	66	77	96	60	72	2	3	NO	NO	NO	NO
25	130	86	101	120	86	97	124	86	99	120	76	91	112	68	83	114	64	81	110	66	81	108	68	81	106	64	78	2	2	NO	NO	NO	NO
26	136	90	105	130	84	99	126	80	95	116	78	91	112	74	87	110	72	85	100	70	80	96	66	76	92	62	72	2	3	NO	NO	NO	NO
27	132	92	105	118	85	96	120	86	97	116	82	93	114	78	90	80	74	76	100	68	79	96	64	75	92	60	71	2	3	NO	NO	NO	NO
28	140	88	105	136	84	101	132	80	97	126	76	93	120	72	88	116	70	85	112	66	81	104	62	76	98	58	71	2	2	NO	NO	NO	NO
29	126	90	102	128	86	100	120	82	95	116	80	92	112	76	88	108	74	85	104	72	83	100	68	79	96	64	75	3	3	NO	NO	NO	NO
30	130	88	102	130	90	103	126	84	98	120	80	93	114	74	87	110	68	82	106	64	78	100	60	73	96	56	69	2	2	NO	NO	NO	NO
31	126	88	101	124	86	99	116	84	95	116	80	92	112	78	89	108	74	85	104	70	81	98	66	77	94	66	75	2	3	NO	NO	NO	NO
32	134	92	106	130	90	103	126	90	102	122	88	99	116	84	95	114	80	91	110	74	86	112	70	84	104	66	79	2	3	NO	NO	NO	NO
33	128	90	103	130	94	106	126	90	102	124	88	100	116	84	95	114	78	90	108	64	79	100	62	75	96	60	72	2	2	NO	NO	NO	NO
34	138	86	103	136	82	100	130	80	97	126	76	93	124	72	89	120	72	88	116	68	84	110	65	80	100	60	73	1	3	NO	NO	NO	NO
35	126	90	102	130	88	102	126	84	98	120	80	93	118	78	91	112	74	87	110	72	85	106	68	81	102	66	78	2	2	NO	NO	NO	NO
36	136	90	105	134	90	105	132	86	101	128	80	96	124	76	92	120	70	87	116	68	84	114	66	82	110	60	77	2	3	NO	NO	NO	NO
37	134	84	101	132	80	97	128	76	93	122	76	91	118	72	87	116	70	85	110	66	81	106	62	77	98	58	71	2	3	NO	NO	NO	NO
38	142	94	110	140	90	107	130	86	101	126	83	97	120	80	93	116	76	89	112	71	85	108	68	81	104	64	77	2	2	NO	NO	NO	NO
39	126	86	99	130	84	99	124	80	95	120	76	91	115	72	86	110	68	82	106	65	79	100	60	73	96	56	69	2	3	NO	NO	NO	NO
40	134	88	103	130	86	101	126	82	97	120	78	92	116	74	88	114	72	86	110	70	83	106	68	81	104	66	79	2	2	NO	NO	NO	NO
41	135	88	104	134	86	102	130	84	99	126	80	95	120	76	91	115	76	89	110	72	85	105	68	80	100	66	77	2	3	NO	NO	NO	NO
42	126	86	99	120	84	96	116	80	92	112	76	88	108	74	85	106	70	82	102	68	79	98	64	75	92	60	71	3	2	NO	NO	NO	NO
43	138	88	105	136	88	104	130	82	98	126	78	94	120	72	88	118	70	86	114	68	83	110	64	79	112	60	77	2	3	NO	NO	NO	NO
44	124	86	99	122	84	97	120	80	93	116	76	89	112	72	85	106	68	81	102	66	78	96	63	74	90	56	67	1	3	NO	NO	NO	NO
45	140	84	102	136	80	98	130	80	97	128	76	93	122	72	89	118	74	89	114	72	86	110	70	83	102	66	78	3	2	NO	NO	NO	NO
46	124	84	97	120	80	93	116	78	91	112	74	87	110	72	85	104	68	80	100	65	77	98	64	75	94	60	71	2	3	NO	NO	NO	NO
47	130	86	101	120	86	97	124	86	99	120	76	91	112	68	83	114	64	81	110	66	81	108	68	81	106	64	78	2	2	NO	NO	NO	NO
48	140	88	105	136	84	101	132	80	97	126	76	93	120	72	88	116	70	85	112	66	81	104	62	76	98	58	71	1	2	NO	NO	NO	NO
49	134	92	106	130	90	103	126	90	102	122	88	99	116	84	95	114	80	91	110	74	86	112	70	84	104	66	79	3	2	NO	NO	NO	NO
50	136	90	105	134	90	105	132	86	101	128	80	96	124	76	92	120	70	87	116	68	84	114	66	82	110	60	77	2	3	NO	NO	NO	NO

LIGNOCAINE																										
S NO	NAME	AGE	SEX	IP NO	BMI	TYPE OF SURGERY	HEART RATE (In Minutes)										BLOOD PRESSURE (In mm Hg)								
							BEFORE EXTUBATION		DURING EXTUBATION	AFTER EXTUBATION							BEFORE EXTUBATION						DURING EXTUBATION		
							0	1		1	3	5	10	15	20	25	30	0 Minute			1 Minute					
																		S	D	M	S	D	M	S	D	M
1	Palraj	40	M	79940	24.3	Lap cholecystectomy	82	86	105	102	97	91	88	80	78	74	72	118	80	93	120	90	100	130	98	109
2	Balkees	30	F	41711	22.2	Lap appendicectomy	90	96	110	112	108	105	96	92	88	84	82	124	84	97	128	88	101	136	100	112
3	Bishana	40	F	5757	22.7	Lap appendicectomy	86	90	114	118	120	108	103	94	90	85	82	130	90	103	140	98	112	150	100	117
4	Manjunathan	26	M	10083	20.8	Lap appendicectomy	76	82	104	110	108	98	92	86	83	80	75	124	80	95	128	84	99	136	94	108
5	Lakshmi	30	F	17852	24.9	Lap appendicectomy	84	90	112	116	120	114	103	95	90	84	82	128	84	99	132	88	103	142	100	114
6	Kandasamy	45	M	39128	19.2	Lap appendicectomy	80	88	104	100	96	90	82	74	70	66	58	124	70	88	130	78	95	140	94	109
7	Jothimani	45	M	17775	23.8	Lap cholecystectomy	70	78	94	98	92	88	85	80	72	65	60	120	74	89	128	82	97	140	96	111
8	Nadhiya	30	F	72	22.2	Lap appendicectomy	96	100	118	124	128	118	112	108	102	95	90	124	82	96	130	86	101	142	96	111
9	Mohanraj	34	M	43218	20.6	Lap cholecystectomy	75	78	94	98	92	88	85	80	76	70	66	118	76	90	126	80	95	138	90	106
10	Faritha Banu	39	F	44387	23.1	Lap appendicectomy	80	84	98	106	110	104	92	86	80	75	68	120	80	93	124	84	97	136	94	108
11	Mariyammal	45	F	4161	19.4	Lap cholecystectomy	64	72	90	96	95	92	86	84	80	75	70	126	86	99	132	92	105	146	100	115
12	Sudha	31	F	40214	24.1	Lap cholecystectomy	76	86	104	110	102	90	84	78	72	66	62	130	74	92	134	80	98	142	94	110
13	Vasantha Kum	21	M	47139	22.5	Lap appendicectomy	84	90	112	116	114	108	102	92	86	80	76	126	84	98	130	88	102	142	100	114
14	Karthikeyan	41	M	44041	24.8	Lap appendicectomy	90	98	114	122	116	110	108	98	95	90	86	126	80	95	132	86	101	144	96	112
15	Senni	38	M	48220	23.9	Lap cholecystectomy	78	84	100	106	108	100	92	86	80	76	70	132	80	97	138	88	105	150	100	117
16	Juveriya	24	F	83803	22.3	Lap cholecystectomy	96	106	118	122	120	108	102	96	94	90	86	114	76	89	122	82	95	134	92	106
17	Pushparaj	40	M	13614	23.3	Lap appendicectomy	86	92	104	108	100	94	88	85	78	72	65	110	72	85	114	76	89	130	90	103
18	Jothimani	35	F	13083	21.2	Lap appendicectomy	84	90	106	110	114	106	100	96	92	88	80	120	78	92	126	84	98	136	94	108
19	Gomathy	27	F	46157	19.9	Lap appendicectomy	72	78	96	90	86	82	78	74	68	64	62	118	68	85	124	76	92	136	92	107
20	Suseela	40	F	15098	23.8	Lap appendicectomy	90	96	110	112	114	106	100	90	86	82	78	130	80	97	134	86	102	142	94	110
21	Jiaml	30	M	56928	22.3	Lap appendicectomy	86	98	116	120	118	112	106	100	96	92	85	118	70	86	126	76	93	138	90	106
22	Priya	21	F	46410	20.2	Lap appendicectomy	74	80	96	100	104	96	90	88	82	76	70	116	76	89	120	82	95	134	94	107
23	Leo fernandaz	24	M	16567	21.2	Lap appendicectomy	86	98	106	120	124	120	112	106	98	95	90	124	82	96	130	86	101	140	96	111
24	Sripriya	20	F	17116	22.7	Lap appendicectomy	68	78	96	94	92	82	80	75	70	66	62	120	70	87	124	76	92	132	84	100
25	Salim	34	M	16291	24.1	Lap appendicectomy	82	88	106	110	108	100	94	88	84	78	72	120	76	91	128	82	97	138	94	109
26	Madhesh	22	M	57085	22.9	Lap appendicectomy	92	88	108	110	106	98	92	88	85	80	74	120	70	87	126	74	91	138	94	109
27	Dhanalakshmi	35	F	50054	24.8	Lap appendicectomy	70	78	96	100	104	98	92	85	80	76	70	116	80	92	122	84	97	132	94	107
28	Mallika	38	F	18763	21.2	Lap appendicectomy	90	98	114	122	116	110	102	95	92	86	80	120	74	89	124	82	96	132	86	101
29	Vennila	32	F	57210	24.1	Lap appendicectomy	84	90	108	110	115	106	100	96	90	86	82	126	80	95	130	84	99	140	94	109
30	Jayasundaram	37	M	45291	23.1	Lap appendicectomy	76	82	100	106	110	98	95	90	84	80	74	118	72	87	126	76	93	138	92	107
31	Jasmine	31	F	53090	22.2	Lap cholecystectomy	70	78	98	104	96	90	85	81	78	70	66	110	72	85	118	78	91	130	90	103
32	Muthu	43	F	27975	23	Lap appendicectomy	92	100	116	120	118	112	106	95	88	84	78	118	76	90	124	80	95	136	94	108
33	Lakshmi	30	F	78217	24.4	Lap cholecystectomy	78	90	108	112	106	95	90	86	82	75	72	122	70	87	130	76	94	144	84	104
34	Thulasiammal	25	F	22267	20.4	Lap cholecystectomy	86	90	108	112	108	100	96	92	88	84	78	125	86	99	130	90	103	142	100	114
35	Kalyani	45	F	69181	20.8	Lap cholecystectomy	94	100	115	118	112	108	100	95	92	86	82	130	88	102	134	94	107	144	100	115
36	Azharuddin	21	M	23058	20.2	Lap appendicectomy	80	84	98	104	100	96	92	88	82	76	72	114	76	89	120	82	95	134	92	106
37	Rajalakshmi	31	F	25417	24.7	Lap cholecystectomy	80	86	104	102	96	92	85	80	76	74	68	122	80	94	128	84	99	136	92	107
38	Michael	32	M	40828	23.7	Lap appendicectomy	88	96	114	110	112	108	102	95	90	84	80	122	84	97	128	88	101	142	98	113
39	Pavithra	20	F	27884	19.4	Lap appendicectomy	80	85	102	96	92	85	82	76	70	66	62	110	68	82	116	76	89	132	90	104
40	Anandha kuma	20	M	21264	22.2	Lap appendicectomy	76	82	98	102	108	100	90	86	80	76	72	116	70	85	122	76	91	134	90	105
41	Manikandan	39	M	73993	22.9	Lap appendicectomy	70	75	94	98	102	96	90	86	80	76	70	118	80	93	126	86	99	138	94	109
42	Reetamary	21	F	30775	22.5	Lap appendicectomy	85	92	106	108	102	90	86	92	75	72	68	126	74	91	132	78	96	142	88	106
43	Kavitha	32	F	31731	23.2	Lap appendicectomy	98	106	120	124	118	112	106	102	94	92	86	132	86	101	138	94	109	148	98	115
44	Jayanthi	40	F	33311	23.8	Lap appendicectomy	70	78	96	94	86	80	76	74	70	66	62	125	70	88	130	75	93	142	90	107
45	Chinnasamy	42	M	39725	23.5	Lap appendicectomy	82	86	102	100	104	95	92	85	80	75	68	124	76	92	130	82	98	138	92	107
46	Thahira	29	F	48989	20.7	Lap cholecystectomy	68	80	96	108	104	98	95	90	86	80	75	118	74	89	122	80	94	134	90	105
47	Velankanni	33	F	64243	21.6	Lap cholecystectomy	78	86	104	108	102	95	92	86	82	78	74	120	78	92	126	82	97	138	90	106
48	Lakshman	20	M	16548	19.4	Lap appendicectomy	82	88	104	110	106	102	95	92	86	80	75	116	76	89	126	84	98	138	94	109
49	Mujeeb	20	M	4396	20.6	Lap appendicectomy	76	82	98	112	106	100	96	92	86	82	78	122	78	93	128	82	97	138	94	109
50	Geetha	31	F	38938	20.2	Lap appendicectomy	98	102	118	122	118	112	106	96	92	84	80	126	84	98	132	92	105	140	96	111

LIGNOCAINE																													
S NO	BLOOD PRESSURE (In mm Hg)																								EXTUBATION QUALITY SCORE	EMERGENCE AGITATION SCALE	ADVERSE EFFECTS			
	AFTER EXTUBATION																													
	1 Minute			3 Minute			5 Minute			10 Minute			15 Minute			20 Minute			25 Minute			30 Minute					RESPIRATORY DEPRESSION	VOMITING	LARYNGOSP.	BRONCHOSP
	S	D	M	S	D	M	S	D	M	S	D	M	S	D	M	S	D	M	S	D	M	S	D	M			NO	NO	NO	NO
1	136	95	109	128	88	101	136	84	101	132	84	100	124	82	96	118	78	91	114	76	89	110	76	87	2	2	NO	NO	NO	NO
2	140	98	112	136	94	108	132	90	104	126	87	100	120	84	96	118	80	93	114	76	89	108	72	84	3	1	NO	NO	NO	NO
3	148	98	115	144	98	113	136	94	108	130	90	103	126	86	99	120	84	96	117	80	92	112	76	88	3	2	NO	NO	NO	NO
4	140	98	112	136	96	109	132	92	105	128	88	101	120	82	95	118	78	91	114	72	86	108	68	81	3	2	NO	NO	NO	NO
5	148	98	115	140	96	111	134	92	106	128	88	101	124	84	97	120	80	93	116	80	92	110	76	87	2	2	NO	NO	NO	NO
6	144	100	115	138	96	110	130	92	105	126	88	101	120	86	97	118	82	94	112	76	88	108	72	84	3	1	NO	NO	NO	NO
7	142	98	113	136	92	107	130	88	102	126	84	98	120	78	92	116	72	87	110	68	82	104	62	76	3	2	NO	YES	NO	NO
8	146	98	114	144	96	112	136	94	108	130	90	103	128	86	100	124	84	97	120	80	93	116	78	91	2	2	NO	NO	NO	NO
9	136	88	104	138	86	103	130	82	98	126	76	93	120	72	88	116	66	83	108	60	76	100	55	70	3	2	NO	NO	NO	NO
10	142	98	113	140	98	112	136	86	103	130	82	98	124	78	93	120	74	89	116	70	85	112	66	81	3	2	NO	NO	NO	NO
11	142	98	113	136	96	109	132	92	105	128	90	103	124	88	100	118	84	95	112	80	91	106	76	86	3	1	NO	NO	NO	NO
12	140	94	109	140	90	107	135	86	102	130	82	98	126	80	95	120	76	91	114	70	85	102	66	78	2	2	NO	NO	NO	NO
13	146	100	115	140	98	112	134	94	107	126	88	101	120	84	96	118	80	93	114	76	89	108	72	84	4	2	NO	NO	NO	NO
14	146	98	114	140	94	109	136	90	105	132	86	101	126	85	99	122	82	95	116	76	89	112	70	84	2	2	NO	NO	NO	NO
15	146	98	114	140	94	109	134	90	105	128	86	100	122	84	97	116	78	91	112	72	85	110	68	82	3	2	NO	NO	NO	NO
16	140	88	105	136	86	103	130	82	98	126	78	94	120	74	89	116	70	85	110	68	82	106	64	78	3	2	NO	NO	NO	NO
17	130	88	102	126	86	99	120	82	95	116	80	92	110	74	86	102	68	79	96	62	73	90	56	67	3	2	NO	NO	NO	NO
18	140	90	107	136	88	104	134	84	101	130	80	97	124	76	92	120	78	92	116	74	88	110	70	83	3	2	NO	NO	NO	NO
19	138	90	106	132	90	104	126	88	101	120	84	96	118	80	93	112	75	87	105	70	82	100	64	76	4	2	NO	NO	NO	NO
20	146	100	115	140	96	111	136	92	107	132	88	103	128	84	99	122	80	94	116	76	89	112	74	87	3	3	NO	NO	NO	NO
21	144	94	111	140	90	107	135	88	104	132	85	101	126	82	97	120	75	90	115	70	85	112	68	83	3	2	NO	NO	NO	NO
22	138	98	111	136	94	108	130	90	103	126	88	101	122	84	97	116	80	92	112	78	89	106	74	85	3	2	NO	NO	NO	NO
23	136	92	107	130	90	103	126	86	99	124	82	96	120	78	92	115	72	86	112	68	83	108	64	79	2	2	NO	NO	NO	NO
24	135	86	102	130	84	99	126	80	95	122	76	91	116	70	85	110	68	82	102	64	77	98	56	70	3	3	NO	NO	NO	NO
25	140	96	111	136	96	109	132	94	107	126	90	102	122	86	98	118	82	94	110	76	87	106	70	82	3	2	NO	NO	NO	NO
26	138	90	106	134	88	103	128	82	97	122	76	91	118	72	87	110	66	81	102	62	75	96	54	68	2	2	NO	YES	NO	NO
27	136	94	108	132	90	104	128	88	101	120	84	96	115	80	92	108	76	87	102	72	82	98	68	78	3	2	NO	NO	NO	NO
28	144	96	112	140	92	108	136	88	104	130	84	99	124	80	95	120	75	90	115	70	85	110	66	81	3	2	NO	NO	NO	NO
29	140	96	111	136	94	108	130	88	102	126	84	98	120	80	93	118	76	90	112	72	85	106	66	79	3	2	NO	NO	NO	NO
30	136	88	104	130	82	98	126	80	95	120	76	91	116	74	88	112	70	84	106	66	79	100	60	73	2	2	NO	NO	NO	NO
31	130	88	102	128	86	100	124	84	97	120	80	93	116	78	91	110	74	86	106	70	82	100	68	79	3	3	NO	NO	NO	NO
32	140	92	108	136	92	107	130	88	102	128	86	100	122	82	95	118	78	91	114	74	87	110	68	82	3	2	NO	NO	NO	NO
33	142	80	100	135	76	95	130	72	91	126	72	90	122	70	87	116	68	84	112	64	80	108	60	76	3	2	NO	NO	NO	NO
34	140	96	111	135	94	108	128	90	103	124	86	99	120	84	96	114	78	90	108	72	84	102	66	78	3	2	NO	NO	NO	NO
35	146	100	115	140	98	112	135	94	108	130	90	103	125	86	99	120	82	95	118	78	91	112	72	85	3	2	NO	NO	NO	NO
36	136	94	108	132	90	104	128	86	100	126	80	95	120	76	91	116	70	85	110	65	80	102	60	74	3	3	NO	NO	NO	NO
37	134	90	105	130	88	102	126	84	98	120	80	93	116	78	91	110	72	85	106	68	81	100	62	75	2	2	NO	NO	NO	NO
38	142	96	111	140	94	109	136	90	105	130	88	102	126	84	98	120	80	93	116	76	89	112	72	85	3	2	NO	NO	NO	NO
39	130	90	103	126	88	101	122	84	97	118	80	93	112	78	89	106	72	83	100	66	77	95	62	73	4	2	NO	YES	NO	NO
40	136	94	108	132	90	104	128	88	101	126	82	97	120	78	92	116	72	87	110	68	82	100	64	76	3	2	NO	NO	NO	NO
41	134	90	105	130	86	101	124	86	99	118	82	94	110	78	89	106	74	85	102	70	81	98	66	77	3	2	NO	NO	NO	NO
42	138	88	105	132	84	100	128	80	96	122	76	91	118	72	87	110	68	82	102	64	77	96	58	71	3	2	NO	NO	NO	NO
43	146	100	115	142	98	113	135	95	108	130	92	105	124	86	99	120	86	97	115	82	93	110	80	90	3	3	NO	NO	NO	NO
44	140	90	107	136	88	104	132	84	100	130	80	97	126	76	93	122	74	90	116	70	85	112	66	81	3	2	NO	NO	NO	NO
45	140	94	109	140	90	107	136	88	104	132	84	100	128	80	96	122	76	91	118	72	87	114	68	83	3	2	NO	NO	NO	NO
46	144	98	113	146	94	111	140	90	107	136	86	103	130	82	98	126	78	94	120	75	90	114	70	85	3	2	NO	NO	NO	NO
47	136	90	105	132	88	103	128	85	99	124	82	96	120	76	91	116	72	87	112	74	87	106	70	82	3	2	NO	NO	NO	NO
48	136	90	105	132	85	101	125	82	96	122	78	93	116	74	88	112	70	84	106	68	81	102	64	77	3	2	NO	NO	NO	NO
49	140	96	111	135	92	106	130	88	102	126	84	98	120	80	93	116	78	91	112	75	87	108	70	83	3	2	NO	NO	NO	NO
50	136	98	111	132	94	107	128	90	103	122	85	97	116	82	93	112	78	89	110	74	86	102	70	81	3	2	NO	NO	NO	NO